THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—41ST YEAR SYDNEY, SATURDAY, APRIL 3, 1954 No. 14

Table of Contents.

THE MODE OF ACTION OF CONVULSIVE TREATMENT.

By JOHN BOSTOCK, F.R.A.C.P., D.P.M.,

AND

BERTRAM J. PHILLIPS, M.B., B.S., D.P.M.,

Brisbane.

THE advent of physical methods of treatment has created a revolution in psychiatry resulting in repercussions both in therapy and in administration. Previously, weeks, months or years were necessary to change the psyche, and psychotherapy was (and still is) lengthy and expensive. The limited number of psychiatrists available can never hope to treat by purely psychological means the great number of mental invalids who need our help. On the other hand, many can now be given quick relief by physical methods.

To one acquainted with mental hospitals, general hospitals and private practice as a psychiatrist in the last twenty-five years there is a vivid remembrance of the distressing atmosphere of psychiatric wards prior to the introduction of physical treatments. The change which has taken place is tremendous. Depression, for example, has almost vanished, owing solely to electro-convulsive therapy. The psychiatric wards are now among the most cheerful in the general hospital.

Whilst physical treatments restore to normal many patients previously untreatable, the rapidity of their action also has repercussions on the machinery of mental care. There is created the possibility of treating all but the hopelessly and chronically affected patients outside mental hospitals. This is of utmost importance to the individual, as actively psychotic patients can be saved from the stigma of certification.

The discoveries of Pasteur and Lister led to the creation of the modern efficient hospitals for the active treatment of medical and surgical conditions. The public attitude of despair at entering a hospital was thereby replaced by one of hope. There is no doubt that the discovery of physical methods of treatment is an event of similar significance to psychiatry and to the patient. Already one can see a greater number of patients hopefully entering mental hospitals on a voluntary basis, expecting to be cured.

In spite of this convincing evidence of the value of convulsive therapy, it is found that whilst one psychiatrist treats nearly all his patients with convulsive therapy, another rarely uses it. One of the reasons for this divergence in therapeutic procedure is undoubtedly the diversity of views concerning the *modus operandi*. The purpose of this communication is to place the mode of action on a basis which will allow a more precise evaluation of the

factors which are involved. This, from our experience, undoubtedly creates practical advantages in treatment, since one can turn to good use the side effects of what is in essence a therapeutic chain reaction.

There have been several speculative theories to explain the mechanism of recovery. These, as will be seen, vary from the purely physical to the purely psychological.

Some Current Theories of the Action of Convulsive Therapy.

Meduna (Brain and Strauss, 1945) postulated a biological incompatibility between schizophrenia and epilepsy and so introduced convulsive therapy for schizophrenia. This concept has proved false. It is now recognized that convulsive therapy is of most benefit in the depressive states, and its use in schizophrenia is less certain. Also schizophrenia and epilepsy can exist together in the same patient.

Batt (1943) quotes the suggestion of Fetterman that depression is a downward course in mood with an inherent tendency to reverse itself after a varying interval. The unconscious phase of the treatment is supposed to bring about this reversal and so hasten recovery. Flescher attributes the result to the seizure itself and not to the loss of consciousness. He thinks that it is a means of unloading, in a socially harmless manner, the large amounts of energy directed towards death.

Grinker is credited with saying that in "Metrazol" convulsions the diencephalon is "exploded". He states that the amnesia which follows the convulsion may be considered as a disruption of association pathways.

Abse (1940) suggests that fear plays a large part in convulsive treatment, and he is supported by Rankine Good (1940), who states that "Cardiazol" achieves results through fear produced by the aura preceding the fit. With electrically induced convulsions, however, rarely is any fear remembered by the patient, and the punitive effect is minimal.

With our technique so little fear has been induced that we consider it must play a very small part in the clinical results. Cook (1940), as a result of statistics, reaches a similar conclusion and quotes an interesting experiment performed by Cohen. States of fear were induced in 19 patients by subconvulsive doses of "Cardiazol" for ten daily treatments. After a lapse of one month 16 patients who had not improved were given ten daily convulsive seizures induced by "Cardiazol". Clinical results showed that convulsion therapy was superior to the induced fear technique.

It has been pointed out that surgical treatment by leucotomy has thrown light on the *modus operandi* of convulsive treatment, since the therapeutic results of incising the frontal lobes are in some respects similar to those obtained by passing electricity through the same region. It was thought that the benefit arose from severance of the fronto-thalamic pathways. Owing to technical difficulties, leucotomy lacks anatomical precision. Deductions from its use are therefore limited. Nevertheless, it is important to note that interference with neuron pathways by physical means has outstanding and predictable psychological effects. It suggests that an explanation of the results of convulsive treatment or leucotomy must involve the triad anatomy, physiology and psychology. We believe that it is necessary to accept the concept that there is a physical component to mental activity. It is beyond the scope of this paper to enter into a discussion of mind-body relationships. There is abundant evidence that physiological functioning is associated with mental activity.

The Physiological Aspects of Mental Activity.

The strongest evidence that mental activity has a physical component is that the cerebral neurons must be functioning for consciousness to be present. This is well illustrated in anæsthesia. When the cerebral cortex is sufficiently poisoned, conscious mental processes disappear and probably unconscious mental activity ceases as well. Other factors interfering with neuron functioning influence mental activity. Some agents have permanent effects on thinking through their action on the neurons (for example, chronic infections and injuries), while some others, such as alcohol or anæsthetics, have only a temporary effect. Over-emphasis of the psychological viewpoint has resulted in neglect of the physiological aspects of thinking. This over-emphasis has been well illustrated by a statement of Bernard Hart (1946):

> It is of the utmost importance that, in the final "laws" obtained by either the physiological or psychological conceptions, there should be no mixing of the terms. The physiological laws must contain no psychological terms, and the psychological laws must contain no physiological terms. Nothing but hopeless confusion can result from the mixture of "brain-cells" and "ideas". The reader must be asked to accept this statement as a dogma.

Whilst such assertions may be defended on the plea of expediency in specialized teaching, they are completely untenable in a scientific discussion of treatment of mental disorders by a physical agent. There are many who agree with this view, and some authors postulate an anatomical basis for the physiological aspects of mental processses.

The Engram Theory.

As man becomes conscious of an "idea", impulses travel along certain nervous pathways. It has been postulated that these pathways become "facilitated" by repetition, which is a part of experience. They are called "engrams" (Gordon *et alii*, 1936). There is evidence that the cortical and thalamic regions are chiefly involved. The brain works as a whole, and when a simple "idea" appears in consciousness impulses traverse many areas and association pathways. Impulses to and from the thalamus probably are the physical basis of the emotion accompanying most ideas. Few ideas are in consciousness at a given instant. As various engrams "light up" in orderly sequence among the myriad cells and conducting fibres, there arises in consciousness the train of thought.

A train of thought has no breaks in continuity. The process of thinking is a continuous activity. Association of ideas is somewhat similar to Pavlov's conception of chain reflexes (Frolov, 1938).

Engrams may be conscious, yet it seems that there are subconscious engrams which produce conscious emotion capable of influencing behaviour. An example of this is seen with the "impulse". A person feels an impulse to do something but there has been no reason for it in consciousness. The unconscious engrams can be activated by a commonplace association, and though the patient does not know what is happening, varying emotions may result. Disturbing emotions in psychoneurotic patients may be caused by these subconscious engrams.

It is possible to visualize the physiological aspects of thinking. As experience is gained, engrams are laid down in the nervous system. The recently acquired engrams can inhibit or reinforce engrams acquired earlier in life. This results in the formation of conscious and unconscious engrams. Engrams, with their emotional accompaniment, play their part in the mental processes of our patients.

The concept of a physical aspect to thinking implies the use of energy which now must be considered.

The Hypothesis of the Neural Energy Constant.

The broad principles of the passage of energy in the nervous system have been known for many years.

In 1931 the hypothesis of a "neural energy constant" for cerebral functioning was outlined in the following terms by Bostock (1931):

> 1. The amount of neural energy employed in the neural circuit which subserves consciousness, in a condition of health, is kept at a constant level of intensity and amount, under normal working conditions. This is the "Neural Energy Constant".
>
> 2. The number of neurones in functional apposition in each moment of consciousness is relatively small compared with the total of cells available.

A concept which fixes neural energy of conscious thinking within narrow limits is of great importance, in

that it brings psychological processes within the framework of physiological function. The term "neural energy constant" does not imply invariability. It presupposes constancy within certain bounds, as in body temperature.

The implications of the concept are important. In ordinary behaviour, fatigue of the cerebral mechanism is minimal. Comparatively few neurons are in action at one time. There is sufficient reserve in the engram formation to permit adequate rest for each individual neuron.

When a person deals with new tasks involving concentration, mental fatigue with its attendant symptoms rapidly develops. This is due to the insufficiency of neurons capable of dealing with the new situation. They are used too frequently within the time limits necessary for recuperation and therefore suffer from fatigue. It has been suggested that fatigue states develop in neuroses in a similar manner.

Engram functioning has been likened to the workings of a railway system. One line may be used in several routes or paths. A multiplicity of engrams implies a multiplicity of routes. There is efficiency when the routes are so numerous that there is a sufficient time limit between the passage along individual tracts.

Neuronal fatigue is far more important than is usually realized. Overwork, over-worry and insomnia constitute a formidable triad in nervous invalidism. Each entity creates fatigue.

In the brief space available it is impossible to outline all implications of the neural energy constant. It must be stressed that the concept describes an automatic control of the extremes of consciousness. In good health an energy regulator is all-important. Its role in disease and treatment is equally marked; but before this is dealt with in more detail it is necessary to discuss the anatomical pathways through which the neural energy flows.

Anatomical Pathways.

It is interesting to visualize the extent of an engram. There is much evidence that both the cortex and the thalamus are involved.

Many years ago Henry Head (1920) found evidence that not only pain was appreciated in the thalamus, but that some emotions had a similar location. There is clinical evidence to support such a view. Freeman and Watts infer that the euphoria following prefrontal leucotomy is due to interference with fibres of the fronto-thalamic pathway. After leucotomy, thoughts which were previously disturbing no longer cause anxiety or depression, as the engrams concerned no longer influence the thalamus. Similarly, cauterization of the medial dorsal nucleus of the thalamus, as in thalamotomy, reduces the appreciation of anxiety and depression.

Head also believed that there is a third entity of "awareness" centred in the thalamic region, and recent evidence has demonstrated a "centre" for consciousness in the midbrain. Awareness is the primitive consciousness and is well demonstrated in birds, which have little cortical tissue but well-developed basal ganglia.

It can therefore be assumed that the components of the engram include the cortex, the thalamus and the midbrain.

The Physiological Results of Convulsive Therapy.

There have been few published reports of the changes in the brain due to convulsive therapy. Although probably millions of treatments have been given, few deaths have been reported and autopsy material is scarce.

Granular degeneration of cells and petechial hæmorrhages (Critchley, 1943) have been seen in the cerebral cortex. Undoubtedly all changes are not revealed by histological methods. It is probable that many of the changes are reversible and vanish rapidly after the fit is over, but tend to persist with repeated fits. From clinical observations, the cells and/or synapses which are the physical basis for the more recently acquired and more complex functions of the brain appear to be affected first. Patients can be given a course of convulsions in such a way as to take them through "descending" states of confusion, as in anæsthesia.

When an anæsthetic poisons the cells of the cerebral cortex to produce unconsciousness, it is done in accordance with Hughlings Jackson's "law of descending paralysis" or law of dissolution (Clark, 1937). This law states that when factors interfere with brain function, the more recently acquired and more complex functions of the brain are the first to be affected. The most recently acquired and most complex of cerebral activity is that concerned with judgement and reasoning. Experiments with alcohol give an opportunity to study this law; many states can be seen, varying from mild confusion with impaired judgement, through disorientation, to complete unconsciousness. The normal inhibition of the cruder and simpler patterns of behaviour is inactivated by the alcohol, and the older engrams and even the basic instincts may be released from control. Similar phenomena occur with convulsive therapy. The degenerative process seen in the cell is presumed to be caused by the anoxia produced by the convulsion.

With convulsive treatment the confusion soon disappears after the treatment ceases. As only minor memory defects remain, permanent brain changes must be minimal. It can be argued that the granular degeneration has been repaired or that whenever cortical cell damage is permanent, function has been taken over by other cells. Some authors (Karliner, 1948) state definitely that there is not enough evidence to indicate any structural brain change after the usual clinical use of electro-convulsive therapy.

It must be remembered that degeneration and regeneration proceed together during a course of electro-convulsive therapy, and consequently the frequency of convulsions is an important factor in the production of confusion. Six fits given at the rate of one every second day do not usually produce much confusion, but if they are given at the rate of two every day the patient can be confused to the point of disorientation.

The extraordinary recovery which can take place after the excessive use of convulsive treatment is striking. Thus one of our patients developed *status epilepticus* following "Cardiazol" injection. She had nine convulsions and for three hours was controlled only by general anæsthesia. Every time anæsthesia "became light" she "had another fit". Extreme confusion lasted for months. Her condition resembled senile dementia. She has been under observation for some years and is now normal.

In these cases, while it is possible that some cells may degenerate past recovery, most regenerate to the functional level. As full clinical recovery occurs, other cells, in the process of reeducation, apparently take over the function of those destroyed.

As may be expected, the physiological results of electro-convulsive therapy are not confined to the cortex. The autonomic nervous system and the endocrine systems are also involved. The effects of electro-convulsive therapy on the endocrine system have been reviewed by Ashby (1953). There is evidence that electro-convulsive therapy affects the hypothalamus and the pituitary gland. Even when electro-convulsive therapy is given in subconvulsive doses there is an outpouring of adrenal steroids. It has been suggested that electro-convulsive therapy through the pituitary and adrenal glands may affect cerebral metabolism. However, therapeutic trials of ACTH and cortisone have been disappointing, and there is no convincing evidence as yet to give the endocrine system a leading role in the mechanism of recovery with electro-convulsive therapy.

The clinical changes due to convulsive therapy are well known. Confusion and amnesia occur. Euphoria replaces depression. Often conflicts are forgotten. Anxiety is relieved and autonomic side effects may disappear. In a large percentage of cases there is a return to the usual personality. The reasons for this will now be discussed.

The Mechanisms Underlying Clinical Improvement of Patients.

Since the introduction of the electric method of inducing therapeutic convulsions other methods have been largely discarded. In our discussion on the clinical improvement of patients undergoing classical electro-convulsive therapy, the mechanisms involved in recovery can be classified as follows: (i) the action of electro-convulsive therapy on the engrams; (ii) changes at the thalamic level; (iii) secondary psychological results, based on (*a*) amnesia, (*b*) confusion, (*c*) abreaction, (*d*) release of instincts, (*e*) increased accessibility to psychotherapy.

These factors will now be studied in more detail.

Action of Electro-convulsive Therapy on the Engrams.

In the mentally ill patient the "neural energy" flows within fixed pathways, and this limits his thinking to a fixed pattern. Constant preoccupation with his symptoms—that is, hallucinations, delusions, psychosomatic symptoms *et cetera*—interferes with normal thinking. We consider that convulsive therapy removes "emotional inhibition" and allows "energy" to flow into other pathways. No longer does the energy in conscious thinking activate the same abnormal engrams. It now flows into other pathways resulting in normal thinking. As the most recently acquired engrams—that is, abnormal engrams—are the first affected by convulsive therapy, the liberated energy is relegated to those which have been laid down earlier in life. This is in keeping with the law of Hughlings Jackson, which the following case illustrates.

A female patient was admitted to hospital confused and negativistic, and was given convulsive treatment. On waking from the third treatment she was cooperative and "almost normal", but had relapsed by the following morning. After the fourth treatment she was "normal" and progressed from that time.

We suggest that the events which precipitated this patient's psychosis were of recent origin and the engrams were accessible to three treatments. But as some of the effect of convulsive treatment passes off in a few hours, recuperation of the engrams concerned was possible, and this reactivated the psychosis. The fourth session of electro-convulsive therapy permanently inactivated the offending engrams, thus allowing the neural energy to flow into pathways not concerned with psychotic thinking.

In some cases poor results are obtained with electro-convulsive therapy. It is usually found that in these the illnesses are chronic and the engrams concerned are not accessible to convulsive therapy. There should be no claim that convulsive therapy is a "cure-all". Insulin therapy, prefrontal leucotomy or psychoanalysis may be needed for a satisfactory adjustment of the patient.

Changes at the Thalamic Level.

The disruption of abnormal engrams, which were automatically sending impulses to the thalamus and producing an unpleasant affect, creates a situation in which there is a gradual disappearance of the disturbing emotions of depression and anxiety. The patients become positively cheerful, and their behaviour is in contrast to their previous attitude of disinterest and apathy. As thought and action are greatly dependent upon the emotion of the moment, the results of convulsive therapy are usually pronounced. It is generally agreed that emotion is the compelling force behind action; so it is fortunate that convulsive therapy dispels depression and anxiety and produces euphoria.

It seems that with electro-convulsive therapy some of the neurons of the frontothalmic pathways are not functioning, because the old events, even if "remembered", cannot initiate conduction of impulses to the thalamus and produce anxiety or depression. This is often seen clinically. A distressing event, which prior to convulsive therapy produced disturbing emotions, no longer does so, although the details are well remembered. A similar state exists after prefrontal leucotomy.

Often the origin of a disturbing emotion is unrecognized. It is probably caused by activation by association of engrams which are not appreciated in consciousness, but when stimulated produce anxiety or depression. An example of this is seen where emotion is produced by a few bars of music, flowers, or a perfume associated with some forgotten experience. Unconscious engrams may produce disturbing emotions in the same way.

As convulsive therapy is not selective in its influence, it is not surprising that subconscious causes of emotion are accessible to treatment. The depression or anxiety disappears, irrespective of its origin. With this type of treatment it is interesting, but not essential, to delve into all the causes of mental illnesses.

Convulsive therapy produces changes in functioning of the autonomic system. The psychiatrist has long realized that psychoneurotic patients may suffer from symptoms psychosomatic in nature yet interpreted by the patient as due to organic disease. These symptoms are produced by disordered emotion and often disappear with convulsive therapy.

Patients with headaches of cerebral tumour intensity, gastric disturbances mimicking peptic ulcer, palpitations or functional diarrhœa may lose these symptoms after two or three convulsive treatments. Whereas the symptoms had been so vividly conscious as to form the chief topic of every conversation, they are not mentioned after treatment and may even have been forgotten. This is due to the disruption of engrams previously the cause of disturbing autonomic accompaniments.

It is known that the autonomic nervous system has a cortical representation (Fulton, 1943). Thus stimulation of certain cortical areas can produce cardio-vascular reactions, vasoconstriction or vasodilatation of the extremities and so on. Conditioned reflex salivation and gastric secretion also depend on the integrity of the cerebral cortex. There is also a functional apposition between thalamus and hypothalamus. It is not surprising that convulsive therapy, which acts on cortical cells and produces changes in emotional tone, can cause the disappearance of psychosomatic symptoms.

Secondary Psychological Results.

When a patient is receiving convulsive treatment two processes are proceeding at the same time. One is a disintegration, the other an integration. In the disintegrating process cortical cells are altered and engrams are temporarily or permanently disrupted. This produces destruction of recent engrams and release of older ones. At the same time the psychiatrist, by psychotherapy, tries to build new socially acceptable engrams and to revive useful older pathways.

The disruptive processes in the brain are seen clinically in confusion, amnesia for events, disappearance of disturbing emotions, and release of the "neural energy" and instincts. These results of electro-convulsive therapy are put to constructive ends. Often with the aid of psychotherapy a new individual emerges. Such change can be compared with the partial demolition of a building in order that it may be remodelled into a new structure.

The processes which occur in personality rehabilitation are extremely complex and give scope for the interplay of every factor in dynamic psychology. Some of these will now be considered.

Amnesia.

Electro-convulsive therapy is reminiscent of concussion. The patient is "knocked unconscious". On his awakening there is confusion followed by a gradual return to normal. He has a retrograde amnesia for a few seconds prior to the actual switching on of the current.

The production of amnesia is a predominant feature in convulsive therapy. The patient is apt to worry because she cannot remember the age of her children or her coming to hospital. The relatives are concerned because of the forgetfulness for important events. This amnesia is unavoidable with convulsive therapy and should be explained at the commencement of treatment. Amnesia is of importance in treatment. It may enable the patient to

forget both the painful experience which led to the illness and the feelings of despair or lack of interest which inevitably follow in its wake.

It would be theoretically ideal if convulsive therapy could obliterate selectively the experience or experiences which produce the disturbing emotions. In practice this is not possible, as other events are obliterated at the same time, in accordance with Hughlings Jackson's law. It must be conceded, however, that mental disease is usually the result of many factors, and a single experience is rarely the cause. In many cases the causes are not found by the psychiatrist even after many interviews. He has, therefore, reason to be grateful for "gunshot amnesia".

Forgetting is a normal and essential process. The well-integrated individual can forget his business when he leaves for home, his children when at work, his worries on a holiday. The neurotic, however, does not forget. Often insomnia denies him the normal relief of a nightly amnesia through sleep. The amnesia which follows convulsive therapy banishes the fear of sleeplessness, and sleeping habits become far more regular. The beneficial effects of rest can then be seen clinically.

Confusion.

Production of confusion is an important factor in the action of convulsive therapy. As in alcoholic intoxication, electro-convulsive therapy reduces "inhibition" and self-criticism, revealing the underlying personality. The patient, unaware of his limitations, may become cheerful and interested in the environment, though occasionally non-cooperative or restless.

Electro-convulsive therapy confusion ranges from simple lack of judgement to disorientation. It is seen clinically as an inability to associate ideas, and may persist in a slight degree for months. Inability to remember names of people is a common example.

The changes in behaviour are unpredictable. A quiet stay-at-home man who has never smoked becomes voluble, eager for excitement and cigarettes. A staid housewife may become flirtatious. Such examples are numerous. Confusion, by reducing "inhibition", has given the individual a synthetic courage to move freely in contact with his fellows.

Abreaction.

For many years "abreaction" has been a recognized psychotherapeutic measure. If repressed events which have been accompanied by strong emotions are brought to consciousness with release of emotion, the symptoms may be modified and the patient relieved. This process may be accomplished on the conscious level in the course of conversation or analysis. More frequently it is carried out under hypnosis or subcoma doses of an anæsthetic. We are of the opinion that abreaction often occurs after the convulsion with electro-convulsive therapy while the patient is in a confused condition.

Our view on abreaction may be too narrowed by an implied insistence that the patient must exactly relive an incident in order to discharge the emotion. Often after electro-convulsive therapy the patient may behave in a frenzied manner, have prolonged bursts of weeping or behave in some other way common to abreaction by other methods.

Concerning this, Shorvon and Sargant (1947), discussing ether abreaction, quote Pavlov's experience with his trained dogs, whose conditioned responses disappeared after the shock of being almost drowned. They infer that much of the efficacy of ether abreaction is due to the vivid and generalized emotional state which accompanies the use of the drug.

Whether or not abreaction takes place during and immediately after the fit, there is no doubt that it occurs during the period when consciousness is returning. Struggling, sweating, crying and other signs are often present. The whole picture is that of an abreaction with its autonomic accompaniments.

Release of Instincts.

Psychological health depends largely upon the successful exercise of the instincts within the rules enforced by society. The adequate exercising of an instinct has an alterative effect, which, though difficult to describe, is of tangible proportions. As an example, the satisfaction of hunger by a well-cooked dinner produces a contentment which cannot be explained by the mere intake of calories. It is common knowledge that the correct exercise of the sex and herd instincts produces similar results.

With electro-convulsive therapy the sex instinct is released from inhibition, which results often in flirtations between male and female patients. Open wards, good supervision and mixing of the sexes under hospital conditions make this a safe therapeutic procedure. If properly supervised it may be beneficial, because it diverts the patient's thoughts from his troubles.

A release of the herd instinct is very noticeable. Play and social intercourse soon replace the neurotic's inertia. This has excellent mental and physical effects.

Increased Accessibility to Psychotherapy.

As has already been mentioned, the normal individual has no impenetrable barriers to the acceptance of new ideas. He is open to reasoning and is prepared to alter opinions or actions. He has insight, foresight and adaptability.

The mentally ill patient is extremely inaccessible. He often finds it impossible to change his own viewpoint, even though he may derive some benefit by so doing. Much of the history of psychiatry centres round attempts to break through this inaccessibility to psychotherapy.

Psychoanalysts, hypnotists and persuasionists may spend many hours of work on single individuals; so techniques such as narcosynthesis, ether abreaction and hypnoanalysis have been designed to accelerate the process. All such methods are time-consuming and expensive. Their scope is limited by finance. As a result, few psychiatrists have been satisfied, and most have felt the need for a more rapid means of inducing accessibility to treatment. Electro-convulsive therapy has in this respect added to our psychotherapeutic armoury.

It cannot be too strongly emphasized that electro-convulsive therapy is not the sole factor in the treatment of mentally ill patients. It prepares the ground for psychotherapy, but is not an end-treatment. This is illustrated by the number of patients who relapse after temporary improvement with convulsive therapy. Their fundamental outlook on life has not been changed.

Psychotherapy must be vigorous to correct the patient's morbid thinking, and the environment must be suitably adjusted on his discharge from hospital. Often this ideal is unattainable, owing to the poor personality of some patients or to a hopeless environmental situation.

Let us never regard electro-convulsive therapy as a builder of personality. Electro-convulsive therapy temporarily disintegrates personality, leaving us the opportunity to reform it by psychological reeducation.

The accessibility to suggestion and persuasion in patients is often over-estimated. The physician considers that because the patient has insight and can converse freely this denotes therapeutic accessibility. This is by no means always the case. Sometimes those with the most insight are the most difficult to treat. The suggestionists try to overcome the difficulty by constant repetition or the use of hypnosis. The persuasionists require many hours of argument, all too frequently without visible effect.

The psychoanalyst, whilst avoiding the use of suggestion and persuasion, has evolved a technique of free association aimed at reactivating old engrams which have a high emotional value. Adolescence and childhood are explored by the analyst to find vivid episodes. The life situations of the past are again mirrored in consciousness. The process might be termed one of reeducation at depth.

Electro-convulsive therapy assists all these psychotherapeutic measures by making the patient more acces-

sible. Rigid resistant engrams are broken down. In this receptive state the patient can profit fully from the psychotherapeutic value of suggestion, persuasion and reeducation. He also responds to the beneficial influence of a new environment.

Whilst an adverse environment may not be an invariable cause of psychosis, it is frequently an important precipitating factor. This has been recognized through the centuries. A change, a holiday or a tour has always been a therapeutic standby to the physician. New scenes often create a new interest and a new outlook on life. Electroconvulsive therapy renders the patient more accessible to the beneficial influences of a "change". Whereas before electro-convulsive therapy his interest was centred round his illness, he can now fully appreciate his environment with beneficial results.

It is fortunate that electro-convulsive therapy is usually given in hospital. In most cases the environment from which the patient comes is associated with the immediate cause of the illness. Freedom from home stimuli in itself enables the patient to "forget" or inhibit disturbing thoughts previously kept active through daily contact with their source.

In our experience, except in favourable circumstances, the results of electro-convulsive therapy *plus* hospitalization are better than those of electro-convulsive therapy in the home. It is rare to find relatives with sufficient understanding to carry out the crudest of psychotherapy. In hospital the patient can benefit from the stimuli of a therapeutic community.

During the early stages of electro-convulsive therapy amnesia interferes with psychotherapy. The patient cannot concentrate and forgets advice. Notwithstanding this, there is an increased accessibility to simple suggestion and persuasion. The patient is ready to listen and to discuss the origin of his nervous disorder. He can be persuaded to take measures appropriate for social harmony. He seems ever ready to accept suggestion.

A common example of the increased accessibility to suggestion is seen in the readiness with which patients after convulsive therapy can be induced to change their occupation, go for a holiday or live apart from their previous environment. For weeks previously the suggestion to change, given by the psychiatrist, has been met with obstinate resistance. After the treatment the patient more willingly cooperates.

It seems that electro-convulsive therapy will also make the patient more accessible to the suggestion and persuasion derived from formal group psychotherapy. In his better frame of mind he will more readily accept the suggestions coming from his fellow patients. He is also more ready to indulge in play.

Our attention was drawn to the virtues of recreational therapy largely owing to an observation of the late Dr. S. E. Jones, who remarked that a game of tennis is therapeutically more valuable to the player and onlookers than a session of planned occupational therapy. Experience bears out this contention. Convulsive therapy produces a condition peculiarly adapted for the enjoyment of play. The urge to play, to laugh, to make friendships, to flirt, to enjoy the day and forget the morrow are prerogatives of the young. The patients are reborn as a result of convulsive therapy.

It is our contention that man is a social animal and mental illness is an antisocial deviation. Intensive social activities are a necessary part of effective therapy.

Conclusion.

In concluding our analysis of the hypotheses concerning the *modus operandi* of electro-convulsive therapy, we must point out that there is no claim for completeness. We believe that we have demonstrated some of the fundamental mechanisms underlying recovery with this treatment. We are aware that the final analysis of underlying principles is not possible until much more is known about the nature of mind and its relationship to brain function. Meanwhile we believe that the concepts which we have outlined will help in understanding the mechanisms of recovery from mental illness with convulsive therapy.

Summary.

1. The *modus operandi* of convulsive therapy is largely explicable by conceding a physiological aspect of "thought". The basis of this is the concept of an engram.

2. Electro-convulsive therapy disrupts engrams in accordance with Hughlings Jackson's "law of dissolution". The more recently acquired engrams are disrupted first.

3. In mental illness the neural energy associated with nervous impulses lacks normal lability, but after disruption of engrams the energy is "liberated" to flow in other engram formations.

4. The "liberation" of neural energy has psychological sequelæ. Whereas conscious thought was restricted during illness to certain topics and symptoms, after treatment thought becomes free and can achieve the lability of normal thinking.

5. The above-mentioned changes create an opportunity for psychotherapy by improving accessibility to suggestion, persuasion, explanation and analysis.

6. Secondary psychological effects are amnesia, confusion, abreaction and release of instincts from inhibition. These create valuable avenues for use in reeducating the patient.

7. The *modus operandi* of convulsive therapy is partly physiological and partly psychological. The final result is dependent upon many interrelated factors. It is stressed that their recognition is of material assistance in treatment.

References.

Abse, D. W. (1940), "The Psychology of Convulsion Therapy", *J. Ment. Sc.*, 86: 95.
Ashby, W. R. (1953), "The Mode of Action of Electro-convulsive Therapy", *J. Ment. Sc.*, 99: 202.
Batt, J. C. (1943), "One Hundred Depressive Psychoses Treated with Electrically Induced Convulsions", *J. Ment. Sc.*, 89: 289.
Bostock, J. (1931), "The Neural Energy Constant", Allen and Unwin, 71.
Brain, W. R., and Strauss, E. B. (1945), "Recent Advances in Neurology and Neuropsychiatry", 134.
Clark, A. J. (1937), "Applied Pharmacology", Churchill Series, 170.
Cook, L. C. (1940), "Has Fear Any Therapeutic Significance in Convulsion Therapy?", *J. Ment. Sc.*, 86: 485.
Critchley, Macdonald (1943), "Electro-convulsive Therapy", in "The Medical Annual", 124.
Frolov, Y. P. (1938), "Pavlov and his School", Kegan Paul, 28.
Fulton, J. F. (1943), "Physiology of the Nervous System", Oxford Medical Publications, 429.
Good, R. (1940), "Some Observations on the Psychological Aspects of Cardiazol Therapy", *J. Ment. Sc.*, 86: 491.
Gordon, R. G., Harris, N. G., and Rees, J. R. (1936), "An Introduction to Psychological Medicine", Oxford University Press, 27.
Hart, B. (1946), "The Psychology of Insanity", Cambridge University Press, 18.
Head, H. (1920), "Studies in Neurology".
Karliner, W. (1948), "Neurologic Signs and Complications of Electric Shock Treatments; Review", *J. Nerv. & Ment. Dis.*, 107: 1.
Shorvon, H. J., and Sargant, W. (1947), "Excitatory Abreaction, with Special Reference to its Mechanism and the Use of Ether", *J. Ment. Sc.*, 93: 709.

THE MORTALITY IN AUSTRALIA FROM INFECTIVE DISEASE.

(Concluded.)

By H. O. Lancaster,
School of Public Health and Tropical Medicine, Sydney.

In this paper the infective diseases of the nervous system are discussed. Then a brief mention is made of a number of other infective diseases not yet treated in my series. Earlier papers have dealt with tuberculosis (Lancaster, 1950*a*), tuberculosis of childhood (Lancaster, 1950*b*), pertussis and acute infective diseases generally (Lancaster, 1952*a*), measles, scarlatina and diphtheria (Lancaster, 1952*b*), bowel diseases and typhus (Lancaster, 1953*a*), tetanus (Lancaster, 1953*b*), and respiratory diseases

and influenza (Lancaster, 1953*c*). The diseases covered in these papers comprise approximately the first section of the sixth revision of the International List, except that I have omitted any mention of syphilis or gonorrhœa, and I have considered some closely related diseases from other sections as, for example, it has been difficult to differentiate encephalitis and brain abscess, or epidemic and non-epidemic influenza, or epidemic meningococcal and simple meningitis. This paper deals chiefly with the infective diseases of the nervous system and with some other infections which are rather less common in Australia than the diseases already treated.

Poliomyelitis Mortality in Australia.

The deaths in Australia from poliomyelitis can be studied only since 1921, for up to that time deaths certified as due to poliomyelitis were assigned, according to the rules of the International List, to a rubric which included neuralgia and neuritis or to a rubric including other diseases of the spinal cord. Only at the younger ages are the mortality rates large enough to be of interest to us in this survey. They are given in Table I.

TABLE I.

The Mortality from Poliomyelitis in Australia.

Period.	Sex.	The Deaths from Poliomyelitis per Million *per Annum* at Ages (Years).						
		0	1 to 4	0 to 4	5 to 9	10 to 14	15 to 24	All Ages
1908 to 1910 ..	M.			Not available.				
1911 to 1920 ..	M.			Not available.				
1921 to 1930 ..	M.	16	24	23	13	10	5	7
1931 to 1940 ..	M.	12	28	25	31	17	11	11
1941 to 1945 ..	M.	8	11	10	10	15	5	5
1908 to 1910 ..	F.			Not available.				
1911 to 1920 ..	F.			Not available.				
1921 to 1930 ..	F.	23	23	23	11	7	3	6
1931 to 1940 ..	F.	18	19	19	17	14	7	7
1941 to 1945 ..	F.	6	7	7	7	15	6	5

The epidemiology of poliomyelitis has been discussed by Burnet (1940, 1952). In many countries there has been a change in the age distribution of cases, so that now the higher age groups are more heavily represented than formerly. This is linked with the general decline in the infection rates in infancy and early childhood, the importance of which has been stressed in dealing with other common infections (Lancaster, 1952*a*, 1952*b*). Owing to the small numbers of deaths from poliomyelitis, a study of the deaths is much less valuable than a study of the infections. The death rates from poliomyelitis suggest that perhaps an undue emphasis is placed on the disease by the laity and the public health authorities generally. Since many epidemiological features of poliomyelitis are still unsolved, it is, of course, a legitimate field for research; but it appears that the emphasis laid on it has distracted attention from some other diseases of importance, such as pertussis and measles, as causes of death or morbidity (bronchiectasis) or hydatid disease. In other words, public health practice might well be directed against some of the commoner diseases in which preventive measures are known to be effective.

Encephalitis.

According to the rules of the more recent revisions of the "International List of Causes of Death", lethargic encephalitis is now to be referred to a rubric among the acute infective diseases; but prior to 1922 it was referred to a rubric, "non-puerperal convulsions". There has also been a less well-defined rubric among the diseases of the nervous system which contains those deaths from encephalitis not definitely certified as lethargic, together with encephalitis due to cerebral abscess. Here, in Table II, I have pooled all these cases, so that under this heading of encephalitis are included lethargic and other forms of encephalitis due to specific infective disease organisms and encephalitis due to non-specific types of infection, such as brain abscess. Even so, this group of diseases has not caused an important proportion of the deaths for any period during the years of my survey. The death rates have remained relatively constant over this time. In Table III I have given the *encephalitis lethargica* deaths separately for each year, and also in another column the deaths by year for all other forms of encephalitis and brain abscess. It remains uncertain whether the *encephalitis lethargica* deaths so diagnosed are in fact due to that disease; but the mortality in Australia seems to be of the same order of magnitude as that reported by the Registrar-General in England and Wales, where there is a check on such certifications. The deaths from encephalitis and brain abscess have not fallen appreciably since the early years of the survey.

Meningitis.

According to the earlier revisions of the International List, deaths due to meningitis, other than tuberculous meningitis, were assigned either to the rubric epidemic cerebro-spinal meningitis or to the rubric simple meningitis. This latter was later subdivided into simple meningitis and non-epidemic meningococcal meningitis. Few deaths were assigned to this last subdivision. They are so few that I have been able to ignore the subdivision. In Table III are given the yearly deaths from the two main types of meningitis. It should be noted, however, that it is only within recent years that bacteriological methods have been consistently used to differentiate the meningitides, so that in the earlier years there would have been deaths from meningitis included in the deaths assigned by the statistician to simple meningitis. This view is strengthened by the observation that there is a tendency for the two types of meningitis to be more prevalent in the same years. Two notable epidemic waves of meningitis have occurred, the first in the years 1915 to 1918, the second in the years 1940 to 1945—corresponding, of course, to the war years, with the consequent herding together of large numbers of adults. Security regulations, however, were

TABLE II.

The Mortality from Encephalitis in Australia.

Period.	Sex.	Deaths from Encephalitis per Million *per Annum* at Ages (Years).									
		0 to 4	5 to 14	15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over.	All Ages.
1908 to 1910	M.	17	3	4	2	6	13	15	31	0	7
1911 to 1920	M.	41	9	13	11	15	25	34	23	31	19
1921 to 1930	M.	72	21	24	26	34	45	49	57	57	36
1931 to 1940	M.	34	12	15	17	19	21	30	31	19	20
1941 to 1945	M.	50	16	10	10	18	22	24	30	10	19
1908 to 1910	F.	13	5	4	4	5	4	7	11	0	6
1911 to 1920	F.	33	9	8	10	11	13	10	15	34	13
1921 to 1930	F.	62	18	19	23	27	35	42	49	23	29
1931 to 1940	F.	25	9	8	11	12	19	29	33	15	15
1941 to 1945	F.	41	12	7	9	15	13	20	15	23	15

different in the second World War, so that the recorded deaths in *Demography*, the official bulletin of the Bureau of Census and Statistics, will not include deaths of service personnel. Accounts of these epidemics may be found in Fairley and Stewart (1916), in Holmes (1941) and in Williams (1942). Taking into account the difficulties of differentiating these various forms of meningitis, I have pooled all the non-tuberculous meningitis deaths to form Tables IV and V. Meningitis is an important disease of childhood. The mortality from this group of causes is highest in the first year of life, but it falls during childhood. The mortality rates are practically constant throughout adult life for any given period. At any fixed age there has been a decline in the death rates, which had begun before the advent of chemotherapy. Moreover, the epidemic over the years 1940 to 1945 occurred in spite of chemotherapy.

TABLE III.

The Deaths in Australia from Certain Infections.

Year.	Number of Deaths.					
	Encephalitis Lethargica.	Encephalitis and Brain Abscess.	Epidemic Cerebro-spinal Meningitis.	Simple or Other Meningitis.	Poliomyelitis.	Erysipelas.
1908	—	24	—	676	—	40
1909	—	21	—	616	—	52
1910	—	39	—	567	—	56
1911	—	62	—	636	—	68
1912	—	61	—	749	—	93
1913	—	69	—	753	—	90
1914	—	74	—	812	—	62
1915	—	71	529[1]	680	—	64
1916	—	62	644	557	—	71
1917	—	95	255	435	—	54
1918	—	73	173	410	—	56
1919	—	109	79	402	—	56
1920	—	116	84	512	—	52
1921	—	166	63	495	—	78
1922	60[1]	176	58	460	42[1]	64
1923	57	148	50	438	30	75
1924	45	115	89	415[2]	26	75
1925	88	137	83	237	58	70
1926	74	149	58	276	39	78
1927	63	96	30	273	26	90
1928	59	135	36	261	52	64
1929	64	159	36	264	61	95
1930	47	116	27	186	37	76
1931	36	93	25	170	49	56
1932	36	89	29	172	96	39
1933	38	95	20	176	21	62
1934	37	104	21	207	31	37
1935	28	103	17	162	36	59
1936	21	90	10	173	21	78
1937	21	90	20	154	115	54
1938	17	83	14	182	159	30
1939	16	74	13	147	27	24
1940	20	90	38	201	33	24
1941	36	96	239	216	25	30
1942	23	111	343	230	34	26
1943	26	128	253	210	28	23
1944	13	96	120	165	22	10
1945	12	71	70	140	76	4
1946	23	72	74	128	99	6
1947	23	79	57	140	22	8
1948	13	94	68	125	55	5
1949	22	88	63	100	86	9
1950	17	99	93	121	117	6

[1] Not available before this year.

[2] After this time, this column contains some cases diagnosed as cerebro-spinal meningitis (non-epidemic), about one or two cases per year.

Leprosy.

Leprosy in Australia is endemic now among the aborigines and the half-castes living under conditions of poor hygiene in North Queensland, the Northern Territory and northern Western Australia. According to Cook (1927), it is probable that there was little or no leprosy in the aboriginal population before the coming of the Europeans. He thinks that Chinese coolies, Polynesian indentured labourers and perhaps some Europeans have introduced the disease from abroad. The disease has caused very severe epidemics among the aborigines, who had had no previous experience of the disease. It is now endemic in the aboriginal population and spreads from them to those Europeans who have close contact with them, and in the European population secondary cases occur among the relatives of the infected. This determines the geographic distribution of the disease; for example, in 1911 to 1920 there were 76 deaths from leprosy in Australia, of which 11 were from New South Wales, two from Victoria, 60 from Queensland, two from the Northern Territory, one from Western Australia and none from either South Australia or Tasmania—which points, according to Cook (1927), to the need for a hot, humid climate for efficient transmission of the disease.

Miscellaneous Infections.

Malaria.

Malaria, in general, has never been an important cause of death in the European population in Australia. The deaths for each period are given in Table VI. There is a high masculinity. If the deaths in this table are analysed by States it is found that only in Queensland are there any great number of deaths of females from malaria; indeed, 58 of the 74 female deaths in the period from 1911 to 1920 occurred in that State. It is well known from epidemiological surveys that the Cairns area of northern Queensland and the Northern Territory are areas in which the climate and other factors are suitable for the rapid transmission of malaria (Ford, 1946). Northern Queensland is the only malarious area in Australia in which there is now a large European population. Deaths of ex-servicemen, especially those returning from the Middle East and New Guinea after the first World War, formed an important proportion of the deaths due to malaria over the years of this survey. There have been a number of severe localized outbreaks associated with mining and agricultural ventures in Queensland and the Northern Territory. In some of these epidemics the half-caste and aboriginal populations have suffered severely, but their deaths are not included in the official statistics. However, the deaths of Asiatic pearlers and seamen have been included in the official statistics. The possibility of confusion in the past of malaria with typhus, typhoid fever and Weil's disease should not be overlooked.

Hydatid Disease.

Hydatid disease in Australia, the clinical features of which have been described by Dew (1925), is still quite an important public health problem. There was much interest in the disease some years ago and it was made compulsorily notifiable in Victoria and Tasmania, but recently there has been but little interest in it. Cole (1945) pointed out that the disease was still of importance in Victoria and that there was gross under-notification, the notified cases in the State being about 73% of the number of deaths. He believed that there were about seven times as many new cases as deaths. Cole pointed out that the deaths from hydatid disease were in excess of those from many of the infectious diseases thought still to be a public health problem. Ferro (1948) has made the same point and maintains that the Australian hydatid rate is equal to the Argentinian rate. He may be quoted:

> *Es interesante anotar, que todos los funcionarios que hasta la fecha consulté, daban su impresión digamos personal, pero nunca podían presentar estadísticas o datos concretos más o menos fehacientes.*

Support may be derived for Ferro's general views by noting the crude death rates in Australia (deaths per million *per annum*) for each of the five periods: for males, 12, 10, 8, 9 and 7; for females, 14, 9, 6, 7 and 5. If one considers the increasing urbanization and the reduced mortality rates at operation, then these moderate declines in the mortality rates must indicate defective public health measures. Ferro (1948) quotes various Australian medical authorities as being satisfied that hydatid disease is no longer a public health problem. This view does not appear

TABLE IV.

The Mortality from Meningitis (All Forms, Excluding Tuberculous) in Australia.

Period.	Sex.	Deaths from Meningitis per Million *per Annum* at Ages (Years).							
		15 to 24	25 to 34	35 to 44	45 to 54	55 to 64	65 to 74	75 and Over.	All Ages.
1908 to 1910	M.	55	47	55	74	84	122	55	156
1911 to 1920	M.	126	84	79	91	83	92	84	182
1921 to 1930	M.	36	32	36	41	42	27	35	75
1931 to 1940	M.	21	14	15	19	20	21	21	35
1941 to 1945	M.	39	28	30	46	58	60	3	65
1908 to 1910	F.	53	34	47	54	33	62	51	133
1911 to 1920	F.	61	38	48	56	52	49	59	129
1921 to 1930	F.	23	17	21	21	29	24	25	53
1931 to 1940	F.	9	10	9	11	12	16	4	23
1941 to 1945	F.	20	16	16	31	45	39	30	44

TABLE V.

The Mortality from Meningitis (All Forms, Excluding Tuberculous) in Australia.

Period.	Sex.	Deaths from Meningitis per Million *per Annum* at Ages (Years).								
		0	1	2	3	4	1 to 4	0 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	1633	1060	601	226	121	514	762	156	101
1911 to 1920 ..	M.	1763	978	488	298	220	503	830	139	90
1921 to 1930 ..	M.	876	475	196	131	127	284	378	68	40
1931 to 1940 ..	M.	465	191	124	87	50	113	188	36	26
1941 to 1945 ..	M.	683	334	204	166	84	201	311	63	38
1908 to 1910 ..	F.	1605	936	459	218	181	456	686	116	54
1911 to 1920 ..	F.	1381	840	352	272	187	418	655	107	72
1921 to 1930 ..	F.	672	318	161	136	80	175	283	56	25
1931 to 1940 ..	F.	336	151	77	60	48	84	137	26	21
1941 to 1945 ..	F.	559	217	163	88	66	136	282	44	25

TABLE VI.

The Mortality from the Less Common Infective Diseases in Australia.

Disease.	Deaths in Australia for the Years.									
	1908 to 1910.		1911 to 1920.		1921 to 1930.		1931 to 1940.		1941 to 1945.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
Malaria	141	25	231	74	209	49	125	24	48	10
Hydatid disease	77	88	258	216	232	175	323	221	122	88
Leprosy	31	2	65	11	46	19	55	17	25	11
Mycoses	2	—	34	12	52	34	63	40	35	20
Cholera nostras	3	2	17	10	11	2	—	—	—	—
Plague	25	2	1	—	52	20	—	—	—	—
Trichinosis	9	3	—	—	—	—	—	—	—	—
Anthrax	8	1	31	6	14	1	4	—	4	—
Erysipelas	71	77	352	314	362	403	228	235	46	47
Other infective diseases	126	27	171	129	143	138	277	278	119	134
Smallpox	2	2	3	2	3	1	1	1	—	—
Purulent infections	179	182	809	767	684	558	534	401	220	168

to be shared by the veterinarians such as Carne (cited by Ferro) or Seddon (1950). Seddon (1950) believes that routine treatment of dogs for tapeworm is not common and that there has been no reduction in hydatid disease in food animals. It may be that with the rise of the country base hospital fewer patients are now sent to the cities for operation, and so the clinical impression has risen that hydatid disease is much less.

In Table VI are set out the deaths in each period for either sex from infective causes, not already treated in detail.

Plague.

There has been little plague in Australia. Cumpston and McCallum (1926) are inclined to believe that it had not been introduced into Australia before 1900. They have given a complete bibliography and details of the epidemics in Australia of the years 1900 to 1909, 1921 to 1922, and 1923 to 1925. Since that year there have been no deaths from plague in Australia.

Small-pox.

As can be seen from Table VI, small-pox has only rarely caused deaths in Australia over the years of the survey.

Cholera Nostras.

Cholera nostras is not to be confused with Asiatic cholera, which has never occurred in Australia, but is probably to be taken as a synonym for dysentery or acute gastro-enteritis.

Trichinosis.

Twelve deaths are recorded in the years 1908 to 1910 as due to trichinosis. However, Mr. A. J. Bearup (personal communication) has not found any references in the Australian literature to indigenous cases of this disease. Johnston and Cleland (1912) have given references to

the diseases caused by helminths in Australia. As the deaths certified in 1908 were mainly of the Australian born, doubts may be raised as to the accuracy of the diagnosis.

Mycoses.

The chief disease under the rubric mycoses is actinomycosis. Cases have been found in every State of Australia.

Anthrax.

Anthrax is a rare disease in Australia in humans, although there are several endemic foci for animals, chiefly the northern tablelands of New South Wales.

Erysipelas.

The total deaths from erysipelas for the various periods are given in Table VI. Inspection of the annual deaths in Table III suggests that the introduction of the sulphonamides accelerated the rate of decline of the death rates from erysipelas.

Other Infective Diseases.

In the category "other infective diseases" I have grouped a great number of diseases not individually important numerically as causes of death in Australia: diseases due to viruses not otherwise discussed already in this series, diseases due to spirochætes (non-syphilitic), diseases due to the helminths, and a rubric that occurs in some of the earlier revisions of the "International List of Causes of Death", "other epidemic diseases". This category is chiefly of interest here to show that the great bulk of the deaths due to infective diseases have now been treated in the discussion of the individual diseases in my series of papers. One point of interest arises: the masculinity in this class in the years from 1908 to 1910 is quite different from that in the other periods. This is due to the occurrence of epidemics in the Northern Territory and Western Australia in 1908 and 1909, chiefly in young adult males. Those affected appeared to be almost entirely Japanese and Indonesians. The deaths were possibly due to beriberi or malaria.

Summary.

The discussion of the mortality from infective disease in Australia is concluded with an analysis of the infections of the nervous system. Poliomyelitis, although so important from a morbidity point of view, has not been important as a cause of mortality, nor have the encephalitides. Meningitis (non-tuberculous) has been an important cause of mortality, especially in childhood, but has declined greatly in importance as a cause of death. For the sake of completeness and to assist international comparisons, leprosy, hydatid disease, malaria, plague, small-pox, *cholera nostras*, trichinosis, mycosis, anthrax, erysipelas and "other infective diseases" have all been briefly mentioned. Only malaria and hydatid disease of these last diseases have been considered in detail. The distribution of malaria has been limited to the extreme north in the civil population. Most deaths reported from other parts of Australia have been among servicemen, or the disease has been contracted elsewhere. Hydatid disease has been regarded in the past as an important public health problem. The present widely held view that hydatid disease is no longer a problem is not supported by statistics or by the opinion of veterinarians.

Acknowledgements.

This paper is published with the permission of the Director-General of Health, Commonwealth of Australia. I have also to thank Dr. Roland Wilson, formerly Commonwealth Statistician, for some tabulations of deaths made for me by his department.

References.

Burnet, F. M. (1940), "Biological Aspects of Infectious Disease", Cambridge University Press.
Burnet, F. M. (1952), "The Pattern of Disease in Childhood", *Australasian Ann. Med.*, 1: 93.
Cole, G. (1945), "Hydatid Disease in Victoria", *Health Bulletin*, Numbers 81 and 82, Department of Health, Victoria.
Cook, C. (1927), "The Epidemiology of Leprosy in Australia", Service Publication Number 38, Department of Health, Commonwealth of Australia.
Cumpston, J. H. L., and McCallum, F. (1926), "The History of Plague in Australia", Department of Health, Commonwealth of Australia, Government Printer, Melbourne.
Dew, H. (1928), "Hydatid Disease", Australasian Med. Pub. Co., Sydney.
Fairley, N. H., and Stewart, C. A. (1916), "Cerebro-spinal Fever", Quarantine Service, Commonwealth of Australia, Service Publication Number 9, Government Printer, Melbourne.
Ferro, A. (1948), *"Difusion de la Hidatidoses", Bol. Secc. Hidatidosis, Minist. Sal. Pub. Prov. Buenos Aires"*, 1: 2.
Ford, E. (1950), "The Malaria Problem in Australia and the Australian Pacific Territories", M. J. Australia, 1: 749.
Holmes, M. J. (1941), "Report on Cerebro-spinal Meningitis", M. J. Australia, 1: 541.
Johnston, T. H., and Cleland, J. B. (1912), "The Helminth Parasites of Man in Australia", "Report of the 13th Meeting of the Australasian Association for the Advancement of Science, 13: 301.
Lancaster, H. O. (1950*a*), "Tuberculosis Mortality in Australia, 1908 to 1945", M. J. Australia, 1: 655.
Lancaster, H. O. (1950*b*), "Tuberculosis Mortality of Childhood", M. J. Australia, 1: 760.
Lancaster, H. O. (1952*a*), "The Mortality in Australia from Acute Infective Disease", M. J. Australia, 1: 175.
Lancaster, H. O. (1952*b*), "The Mortality in Australia from Measles, Scarlatina and Diphtheria", M. J. Australia, 2: 272.
Lancaster, H. O. (1953*a*), "The Mortality in Australia from Typhus, Typhoid Fever and Infections of the Bowel", M. J. Australia, 1: 576.
Lancaster, H. O. (1953*b*), "The Mortality in Australia from Tetanus", M. J. Australia, 2: 417.
Lancaster, H. O. (1953*c*), "The Mortality in Australia from Infections of the Respiratory System", M. J. Australia, 2: 672.
Seddon, H. R. (1950), "Diseases of Domestic Animals in Australia: Part I. Helminth Infections", Service Publication (Division of Veterinary Hygiene) Number 5, Department of Health, Canberra.
Williams, H. (1942), "Meningococcal Infections in Infancy and Childhood, Part I", M. J. Australia, 2: 535.

WAR GASES AND CHRONIC LUNG DISEASE.

By Alan H. Penington,
Melbourne.

Experience is our only teacher in war and peace.
—Walter Savige Landor.

On the generation of servicemen engaged in the conflict of 1914-1918 the impact of noxious gases employed in both offence and defence had immediate and lasting effects, both physical and psychological. On members of this and immediately succeeding generations the impact has been heavy, socially and economically, affecting home life, industry and governmental economy. Apart from these considerations, the physical impact on men exposed to these substances has been a source of interest to, and speculation by, the medical profession, and amidst the emotional and sentimental miasma which may cloud medical opinion, much controversy and many conflicting hypotheses have been nurtured and flourished. Effluxion of time has now solved many of these conflicts, so that in 1953, thirty-five years after the last large-scale use of these substances, some perspective has been obtained, and the permanent or transient nature of the injuries sustained from these gases can be assessed.

The purpose of the present study is to review the circumstances under which the injuries were first sustained, and the evolution of the medical opinions and evidence concerning the possible permanence of pulmonary sequelæ when gas was encountered. The passage of many years has allowed a more scientific appraisal of the effects of these gases, and serves but to confirm the opinion that relatively few servicemen suffered any permanent pulmonary injury from these substances, and that "the use of the asphyxiating shell is far less inhuman than the employment of submarine boats" (Roberts, 1915).

Historical Survey.

The use of toxic gases in warfare was not new in 1915. The Spartans had, in 429 B.C., used fumes from burning sulphur, pitch and charcoal against the city of Plataea

(Thucydides, Book II), and during the Middle Ages the smoke from burning straw and various smokes and vapours from pitch and sulphur were employed during certain sieges. That similar substances might well be employed in any future conflagration was recognized by the Hague Conventions of 1899 and 1907, which passed an Article which bound all participants "to abstain from the use of projectiles, the object of which is the diffusion of asphyxiating or deleterious gases". This Article was not signed by the representative of the United States, but was confirmed by Great Britain, France and Germany.

Despite the agreement of the Hague Convention, the organization of the chemical firms of Germany in the early twentieth century, which had reached a high level of scientific achievement, was of such a nature and so closely linked to research that it permitted rapid production of substances which might be used in warfare. This industry was allied in interest to the German High Command, who could call upon the manufacturing chemists of the dye industry in the synthesis and development of a weapon which might be used effectively in "surprise" attacks and prove the necessary agent to lead to victory.

The British and French scientific bodies, on the other hand, had been engaged in work removed from the production of substances readily converted to warfare, and the initiative for the use of these substances therefore rested with the German forces.

At 5 p.m. on April 22, 1915, at Langemarck, near Ypres, a discharge of chlorine gas in a cloud formation was made from the German lines against French Colonial troops. The surprise was absolute (Butler, 1943), and the troops broke and fled under the cloud of greenish and choking gas. The breach in the lines was not effectively exploited, and on April 23 it was reported by a Canadian medical officer in a unit adjacent to the area that "fortunately the supply was not unlimited and, after the gas cloud passed over, most of the men soon recovered from its effects" (quoted by Butler, 1943). Defensive measures, at first ingeniously devised masks, and later various types of respirators, were rapidly developed with varying degrees of efficacy, and effective surprise was not again obtained. From April, 1915, to August, 1916, "clouds" of gases were employed consisting chiefly of chlorine, and chlorine and phosgene, with an interlude from June to December, 1915, during which tear shells were employed.

The Australian infantry forces first encountered gas on the night of June 16-17, 1916, at Messines, when the Germans sent over a gas cloud containing smoke mixed with a little gas, followed, after an interval, by a second cloud containing phosgene with some chlorine. Fortunately very few Australian units were then in the line, the only unit, the 25th Battalion, having no casualties. The 1st Anzac Corps was further in the rear and the margin of the cloud passed over some units, some members of the corps being slightly affected.

From July, 1916, to July, 1917, the "clouds" of gas having proved an unsatisfactory method of employment, gas shells with fillings of phosgene, diphosgene and chlorarsine were employed. These substances were lethal and dramatic in their effects, but were soon overshadowed by the development and use in July to December, 1917, of the new substance, "yperite", now referred to as "mustard gas". This substance, being liquid and having a very low vapour pressure at 30° C. (86° F.), was persistent and remained lying on the ground, where it contaminated articles and areas of soil. The fumes were most effectively excluded from the respiratory system by the box respirator then employed. "Yperite" or "mustard" is a vesicant, and its effects on the troops were described, in the worst cases, as most pitiful, "eyes swollen and streaming, voices gone, and bodies blistered".

The consummation of the gas warfare was seen, in both offence and defence, between November, 1917, and November, 1918. During this period immense amounts of mustard gas, arsenical smokes, and phosgene were used by the Germans, both in projectiles and from projectors in the form of clouds. During their retreat of September and October, 1918, the Germans used "an unprecedented deluge of Yperite—the gas which denies the position to each side alike" (Butler, 1943).

Between April, 1915, and November, 1918, the substance most extensively used by the German forces was "yperite" or "mustard gas". It was, however, only one of 30 different chemical substances employed, the remainder comprising the lethal gases—chlorine, phosgene, diphosgene and chloropicrine—the tear gases, irritants causing sneezing *et cetera*, and the various smokes employed for differing purposes. The term "gas" without particularization may mean any one or any combination of these substances, and in fact the methods of production were such that very few of the materials employed were chemically pure. Where "gas" is regarded as an ætiological factor in the production of pulmonary disease it is important that the nature of the particular gas should, so far as possible, be ascertained, for much is known of the toxic effects of each of these substances.

Whilst chemical warfare continued, it is of importance to appreciate that the ordinary respiratory infections endemic in any body of men were occurring amongst soldiers. This fact caused much confusion in later years, for it was at times difficult to distinguish between acute respiratory infections and the immediate respiratory effects of some of these chemical substances.

Toxicology.

The physiological actions of the chemical substances employed in warfare have, for the purposes of clinical medicine, been grouped under four major headings, depending on the site of major incidence of their effects. These substances are not in general absorbed into the blood-stream, and their actions are local, so that a valid classification may be made on such a basis.

The four groups comprise the acute lung irritants, the lachrymators, the sensory irritants to the eyes, nose and chest, and the vesicants (Butler, 1943). Some substances may produce two or more effects, and the pharmacological actions may not, in fact, be confined only to one site. Therefore a further classification based on the effects of these gases was devised by Dr. C. A. Courtney, who on August 7, 1933, suggested that a more satisfactory grouping was the following:

1. Pulmonary irritants. The principal pharmacological action of these substances arose from the release of a halogen radical in the pulmonary mucosa, with resultant pulmonary œdema, anoxæmia, and death from asphyxia. Chlorine, phosgene and chloropicrine were the most important of this group. Acute pulmonary irritation was the major feature, though this irritation with consequent severe œdema was not associated with cell necrosis, the effects being confined largely to capillary permeability (Staehelin, 1920).

2. Vesicants. These substances chiefly affected the skin, conjunctivæ and the mucous membrane of the respiratory tract, and did not, as a rule, cause pulmonary œdema. Their effects were due to direct destruction of cells with necrosis of tissues, secondary infection of exposed or involved mucosa being not uncommon. Of these gases, mustard gas was the major example.

3. Lachrymatory substances. These chemicals were irritants of mucous membranes, and their action was particularly on the eyes and nasal mucosa. They had a high atomic weight and were, in the main, crystalline in nature, the halogen radical being pharmacologically active and causing a profuse flow of tears with photophobia. Their effects were transient.

4. The sensory irritants or sternutators. These caused sneezing, irritation of the eyes, nose and throat, nausea and nervous symptoms. No permanent damage was observed, although the pharmacology of these substances has not been fully elucidated. Their effects may be associated with the physical characters (crystalline) of most of these substances.

5. The paralysants. Of these, hydrocyanic acid was a striking example. These substances are direct protoplasmic poisons which act on the nervous system, so that when

they were encountered in high concentrations, death occurred rapidly.

6. The gases which interfered with the oxygen-carrying capacity of the blood. These substances were absorbed into the blood-stream and combined with hæmoglobin to prevent the formation of oxyhæmoglobin. Sulphuretted hydrogen and carbon monoxide were two of these, and recovery from their effects was uncommon.

A similar classification is employed by the British War Office in its "Medical Manual of Chemical Warfare" (1941).

As this study is concerned primarily with the pulmonary effects of the war gases, the immediate effects of these substances on the respiratory tract is important. Those substances described as acute lung irritants — namely, chlorine, phosgene and chloropicrine—were disabling in concentrations of 1:10,000 for chlorine to 1:200,000 for chloropicrine. They caused acute swelling and œdema of the pulmonary mucosa, particularly in the bronchi and alveoli, with reactionary lymphatic or tissue fluid œdema. The associated clinical syndrome was one of "choking, coughing, gasping for breath through irritation and spasm, retching, sternal pain and thirst followed by the phenomena of anoxæmia, weakness and lassitude" (Butler, 1943). It was observed that when recovery occurred the œdema had usually subsided within a few days, and unless infection was superadded the pulmonary condition returned to normal. In some cases, however, associated with the pulmonary effects was a degree of hæmoconcentration which was recognized by the resulting increased cardiac action. The tachycardia which frequently persisted after such "gassing" was, in many cases, associated with the emotional problem engendered by this type of warfare and, as Butler states, "the acute injury inflicted by pulmonary irritants and the chronic lesions left by gas wounds helped to create another major problem of military medicine—the 'effort syndrome'—by superimposing a 'neurosis'".

The effects of concentrations of mustard gas vapour on the respiratory epithelium have been studied in man and in animals by very many workers. Warthin and Weller (1919) demonstrated that the effect of mustard gas vapour on the respiratory mucosa was most severe about "the nose, back of the tongue, palate, pharynx and larynx, decreasing in intensity downwards". Local injury to the epithelium was seen in desquamative inflammatory changes at these sites, and depending on the severity and depth of necrosis, either superficial or to the level of the submucosa, healing occurred either with complete restoration of the mucosa or with scarring and thickening. Symptomatically, involvement of the larynx, trachea and bronchi was associated with persistent cough and signs of bronchial involvement. If secondary infection occurred, purulent bronchopneumonia developed.

Winternitz (1919) makes the following statement:

> Not only does mustard gas, in lethal concentrations, destroy the lining of the entire bronchial tree, but its necrotizing action involves the lung itself. In more dilute form it produces necrosis of the epithelium of the trachea, which, however, becomes less marked toward the finer ramifications of the bronchi.

The severest effects are in the upper portions of the respiratory tract; but should infection be superimposed upon the necrotic effects of the gas, then the accompanying purulent bronchitis and bronchopneumonia may overshadow and eclipse the chemical effects of the gas.

Whilst the changes described are those encountered in acute cases of mustard gas poisoning, repeated exposure to low concentrations or subtoxic doses of the vapour may, by cumulative effects, reach a toxic level. This has been observed in one of the arsenals in America (Brown, 1948) amongst employees engaged in the filling of gas shells. The pathological changes and the clinical features were those described by Winternitz and by Warthin and Weller as arising in acute poisoning, and differed only in the degree of initial exposure and the rapidity with which they arose. In the cases reported from America these symptoms appeared whilst the employees were directly exposed to the low concentrations of vapour, and did not appear after this exposure had been interrupted or terminated. Poisoning in these cases was due to summation of repeated low concentrations, and did not occur after removal of the employee from the contaminated area.

Functional disorders immediately following gassing became extremely common. Butler (1943) states that "the symptoms known as 'gas effects' are only the outward sign of complex occult toxic actions and physiological and psychic reactions". Similar observations were made by British officers, and a clear warning was given by the British War Office (1941) of the extreme danger of neuroses arising where "exposure to gas, often to minimal and barely toxic concentrations, may yet prove the final factor in upsetting a nervous system already breaking down as the result of physical or mental strain". It was because of such observations in France that the problem of management of gas casualties became more complex than would be expected from a knowledge of the efficacy of defence against these chemicals and the pathology of the lesions in the respiratory tract produced by gas.

Consideration of the toxicology of the chemical agents applied and of the pathological changes produced by these agents indicates that when gas affected the respiratory system, such effects were physical in nature and produced immediate damage to the respiratory mucosa. Such damage consisted of changes which varied from swelling and œdema of the mucous membrane without necrosis to actual death of the tissues. When such a degree of damage occurred, symptoms referable to these tissues appeared at that time. According to the severity of the damage, so did the severity of the symptoms vary; but when necrosis of tracheal or bronchial mucosa took place, the studies of Warthin and Weller on both experimental animals and humans showed that severe symptoms were always present, and the clinical observations of medical officers in the field were completely consistent with these observations.

It is established, therefore, that when noxious gases produced any damage to the respiratory mucosa, this damage was evidenced by respiratory symptoms. If the effect of the gas was severe, respiratory symptoms were severe and were produced at the time of or immediately after such exposure. These symptoms and their pathological causes were aggravated by secondary infection; but there was frequently, in addition, a superadded emotional factor which could not always be related to an organic basis. Persistent exposure to low concentrations of gas could result in toxic effects; but when this occurred, the organic and symptomatic picture was similar to that encountered with exposure to high concentrations, and the symptoms did not arise after removal of the subject from the contaminated areas.

Disposal of Gas Casualties in the Field.

The pharmacology and pathological effects of the war gases and the intensive use of these substances by the Germans caused many casualties amongst men serving in the field. It is apparent from the intensity of the campaigns that there were few soldiers engaged in front line service who were not at some time exposed to such substances, at least in low concentrations. The efficiency of the respirator against respiratory gases was of a high degree, and it gave adequate protection when properly employed; but contamination of ground, materials and clothing inevitably meant that some concentration of gas vapour was encountered by most soldiers after removal of the respirator. This did not, in the majority, produce symptoms of such severity as to make them casualties, and from a study of the pathology of these conditions it follows *ipso facto* that the degree of damage to the respiratory mucosa was not severe. Hoarseness was not an uncommon feature and caused no severe disablement, but tracheitis or bronchitis due to gas was incapacitating, and the psychological upset was often severe.

Because of the problem presented by the soldier who alleged that he had been gassed and presented few, if any, signs of such condition, immediate evacuation as gas casualties was reserved for those men in whom the clinical syndromes left no doubt as to the condition. In 1918 "gas centres" were established in army areas, where casualties

were observed and treatment was given for the milder clinical effects of gassing. Not all soldiers suffering from mild gas effects were evacuated, but when severe pulmonary damage had been sustained, evacuation was inevitable and was usually for a prolonged period. Severe criticism was made of prolonged retention of such men in base hospitals, and Colonel McWhae reported (Butler, 1943) that two chief types of patients were discernible: those with severe chronic bronchitic symptoms, and those who had no bronchial symptoms but complained of tiredness, dyspnœa and precordial pain. Colonel McWhae concluded that of the latter group a very large proportion had been only slightly gassed and no organic signs of disease were present.

Until November, 1918, the total number of gas casualties suffered by the Australian Imperial Force—that is, the number of casualties recorded as "gassed"—was 16,819; of these men, 323 died. The total number of casualties due to wounds in the same force and over the same period was 176,893; of these, 53,561 men died. As the reported "gas casualties" comprised all those soldiers admitted to field medical units, it is apparent that the total percentage of casualties due to gas of sufficient severity to require admission to field medical units was, at its maximum in 1918, only 8·46% of all casualties. These figures indicate that severe gassing was relatively uncommon and that severe pulmonary effects were not the general rule after exposure. However, when a soldier was evacuated and reached a base hospital, this was because of either severe pulmonary involvement or those symptoms which became epitomized as "effort syndrome" or as "D.A.H.". In this latter group the pulmonary effects, if any, of the gas had been either minimal or transient, the major features of the syndrome being primarily of psychogenic origin.

It might be reasonably concluded from a study of the management of these casualties in the field that unless there was evacuation from the front line or a consistent history of pulmonary symptoms with cough and hoarseness, there was no reliable evidence of severe pulmonary involvement by gas. Minor degrees of gassing could occur without such symptoms arising; but these produced no significant change in the respiratory mucosa. Exposure over long periods of time to low concentrations of gas could affect the respiratory symptom; but when this occurred symptoms comparable with those caused by more severe exposure arose and were indistinguishable in character from those due to such severe exposure.

Observations during Early Post-war Years.

Before the use of poison gases during the 1914-1918 war it had been contended that this weapon of warfare, whilst causing temporary incapacity and confusion, was associated with a very low mortality rate, and that no permanent ill effects or wounds were sustained by the combatants. The development of some of the newer substances, particularly mustard gas, was not envisaged when these views were expressed, and until 1918-1919 there was a general impression amongst physicians that when recovery took place no permanent sequelæ would result from poisoning with these substances.

In the immediate post-war years it became apparent to the physicians of all belligerent nations that the earlier impressions were incorrect, and the medical literature of the period from 1920 to 1930 is concerned primarily with establishing the fact that, after severe pulmonary involvement at the time of exposure, a proportion of soldiers were left with permanent pulmonary damage. Price (1938) ably summarized the observations of British medical men when he drew attention to the cases of recurrent bronchitis, bronchiectasis and emphysema which appeared to have a clear association with severe pulmonary effects at the time of gas exposure. However, he emphasized in a letter to the Minister for Pensions (confidential papers dated May 22, 1933) that only a comparatively small percentage of gas casualties suffered from such ill effects, and that "the greater bulk of men, including those with minor symptoms, received no permanent damage". He considered that:

> In dealing with the question of the relationship between any disease of the respiratory system and a previous gassing, it is necessary to have evidence of—
>
> 1. health prior to the gassing with particular reference to records of lung trouble, if any;
> 2. exposure to gas;
> 3. nature of gas;
> 4. effects of the gassing with particular reference to signs of lung involvement indicative of serious and permanent damage to the lung tissues;
> 5. an unbroken connection between the date of gassing and the pulmonary disease under consideration.

In America, because the supposed permanent effects of gassing were clinically, radiologically and pathologically indistinguishable from the diseases of chronic bronchitis, bronchiectasis and emphysema which occurred in civilian life, a Board of Medical Officers was appointed in 1926 to investigate the relationship between exposure to war gases and the subsequent development of chronic pulmonary disease. This Board issued its preliminary report as at June 1, 1928 (confidential papers), and concluded that there were patients who exhibited definite residua due to the effect of gases. However, the Board pointed out that in these patients there had frequently been respiratory infections which had contributed to such residua, and that these infections might have been of major importance. The Board reviewed all gas casualties who had been evacuated to field medical units, and the most significant observation was that the major proportion exhibited complete recovery. Meakins (1936) summarized American opinion in the following statement:

> The after-effects are remarkably few considering the vast number of cases which occurred during 1916-1918. They are divisible into two groups, those who depict "Effort Syndrome" and those in whom there were permanent pulmonary lesions. These were essentially due to scarring and infection.

In France, during the years from 1919 to 1925, opinion varied widely between different groups of physicians, many physicians holding that recovery from gas was complete, whilst others maintained that permanent effects were relatively common. By 1925 opinion had become clarified, and when there had been severe respiratory involvement at the time of exposure, Sergent (1925) concluded that permanent sequelæ might occur, although they were not common, and that these residua were seen in the forms of recurrent bronchitis, sometimes associated with emphysema, bronchiectasis, and a group of symptoms resembling those due to pulmonary tuberculosis. However, the most frequently observed result of war gassing was complete and lasting resolution of the condition with complete recovery of the pulmonary tissues.

German physicians were at this time also describing cases in which, after severe involvement of the pulmonary tissues at the time of the original episode, persistent respiratory symptoms with cough, sputum and dyspnœa were encountered. It was concluded that these symptoms were due to scarring of the bronchi and lungs. Staehelin (1920), in referring to these cases, reiterates that these symptoms can be related to war gas poisoning only when there is a history that "an unbroken connection has existed from the time of the poisoning to the present symptoms".

In Australia the pensions aspect of the problem led to very careful studies by medical consultants. During the early post-war years much faith was placed in the infant specialty of röntgenography. Shortly after the cessation of hostilities the widespread use of radiography in the study of the lungs, combined with a naïve belief that minor degrees of a generalized increase in fibrous tissue in the bronchial walls could be recognized in the poor quality films, resulted in a common diagnosis of "pulmonary fibrosis" which was for some years regarded as due to the effects of gas. The late Sir Richard Stawell in 1932 (Butler, 1943) stated that "the evidence of time and a more scientific attitude towards the significance of shadow pictures have led to a considerable modification of this pathological concept". He concluded that the major late effects of gassing were seen in the cardiac neuroses, but that some permanent respiratory damage did occur in a proportion

of cases. However, because recurrent minor respiratory infections apart from the hazards of active service life could lead to the same pulmonary conditions, "continuity of the manifestations of disease" was of the greatest significance in relating any chronic respiratory disease to gas poisoning.

It is apparent that during the years immediately following 1919 it was generally appreciated that chronic bronchitis, emphysema and bronchiectasis could develop as the result of poisoning by war gases. Such pathological sequelæ were to be anticipated when the pathological lesions described by Warthin and Weller were appreciated. However, as bronchial epithelium has astonishing powers of recuperation, the pathological basis of such sequelæ was the scarring and fibrosis in the subepithelial tissues following severe and deep necrosis caused by gas or by subsequent infection.

During the 1930's Stawell made the following comment (Butler, 1943):

> A most remarkable phenomenon of pathogeny, since 1918 has been the birth, burgeoning, efflorescence and decay of the idea that exposure to an atmosphere containing poison gas, even without any symptoms, might result many years afterwards in pulmonary fibrosis with increasing debility and definite degenerative conditions in the bronchi and lung tissues.

Consideration of the conditions of warfare indicates that few men engaged in front line service would not at some time be in a contaminated atmosphere; but when the pathology of the primary pulmonary lesions is considered, it is apparent that these lesions must inevitably produce symptoms at the time of their infliction. If symptoms did not occur, it can be regarded as self-evident that there was no damage to the bronchi; therefore, *ipso facto*, there could be no sequelæ. Whilst Stawell in 1932 regarded this hypothesis as "decayed", it is surprising that in 1952, twenty years later, the hypothesis is still put forward by claimants who are now in their sixth and later decades. There is no pathological basis for such an hypothesis, and the thesis of "occult gassing" should surely be buried in the archives of the past.

Discussion.

Clinically and pathologically, the pulmonary diseases which have been attributed to war gases comprise the syndromes of chronic bronchitis, emphysema and bronchiectasis. The clinical and pathological features of these syndromes differ in no respect from those which occur in any community, whether exposure to chemical warfare has or has not occurred. Neither can any statistical difference be demonstrated between the ages at which these conditions become apparent in soldiers and in civilians. All the evidence that these conditions are due to "gassing" is entirely presumptive and based on our knowledge of the pathology of acute gas poisoning, and on a chronological association between the onset of symptoms, the history of severe gassing with associated pulmonary symptoms, and progress or persistence of these symptoms since the incident stated to be the cause.

Oswald and his associates (1953) have shown that chronic bronchitis is largely a disease of middle-aged men and that there is a fairly strong family history of this condition. Christie (1951) points out that emphysema is usually a disease of middle-aged men with a history of chronic respiratory obstruction due to asthma, chronic bronchitis, bronchiectasis or some other cause. These views express the opinions of all practitioners who have had experience in chronic chest diseases, and the common incidence of these conditions in civil life, and in a particular age-group, and the uniform clinical and pathological pictures irrespective of the mode of onset, serve to negate "gassing" as a cause of these conditions unless a strong train of presumptive evidence is established.

The mythical "hilar fibrosis", diagnosed so frequently in the early 1920's on the basis of poor quality X-ray films, has been shown by effluxion of time to be non-existent, and represented only a iatrogenic condition. When associated with symptoms, the condition was due to bronchitis, it now being appreciated that there may be no radiological evidence of any pulmonary abnormality in simple bronchitis (Oswald *et alii*, 1953).

In recent years the average age of ex-servicemen from World War I is over fifty years, and awareness of the increasing incidence of carcinoma of the bronchus in this age group has inevitably led former soldiers to attribute this condition to exposure to war gases. Statistically there is no validity in this hypothesis, for there is apparently no higher incidence of the condition amongst those who were exposed to gas than there is amongst adult males who have never encountered these substances. Berenblum (quoted by Willis, 1948) demonstrated that mustard gas and various other halogen compounds inhibited the carcinogenic action of hydrocarbons, and it is therefore an incorrect postulate to regard "gases" as carcinogenic agents. These substances are not, therefore, potential causes of a carcinoma of the bronchus which may develop many years subsequent to exposure.

It has been further postulated that the epithelial changes with areas of squamous epithelium sometimes encountered by Warthin and Weller in their studies of the bronchi following gas poisoning could form a nidus for a later developing carcinoma. However, Weller (1953) has demonstrated that areas of squamous-celled metaplasia are commonly found in normal adult lungs, and there is no evidence, therefore, either that this change is due to gas or that it is a potential source of a carcinoma.

When all the evidence over the thirty-five years that have now elapsed since the cessation of hostilities in 1918 is reviewed the conclusion must be reached that there is presumptive evidence that war gases may cause permanent pulmonary damage. However, such presumptive evidence must, as stated by Dr. (now Sir) Geoffrey Marshall, Honorary Consultant to the British Ministry of Pensions in 1949, be dependent upon the following:

(1) evidence that the gassing had produced respiratory symptoms which necessitated hospitalization for an appreciable time;
(2) evidence of respiratory signs and symptoms indicating bronchitis or emphysema within, at most, 5 or 6 years of the gassing and thereafter a continuing history of these signs and symptoms up to the time when a claim is made;
(3) no obvious or more probable alternative etiology.

It is possible that bronchial involvement may have occurred without prolonged hospitalization during the latter stages of 1918, when there was an acute shortage of man power and men were retained in the field unless evacuation was unavoidable; but when bronchial involvement was of such a degree as to lead to permanent damage, contemporary symptoms were of such severity that some period of hospitalization or evacuation was inevitable.

Conclusion.

A study of the nature and history of the toxic gases employed during the hostilities of 1914-1918 indicates that these substances caused considerable immediate morbidity, but that permanent pulmonary injury was sustained only in a minority of those affected by these substances. When permanent damage occurred to the lungs, this became evident as chronic bronchitis, emphysema or bronchiectasis.

Pulmonary damage which left permanent effects was sustained only when exposure was severe, and the immediate pulmonary injury was of such severity as to cause immediate invalidity and evacuation. Only when persistent symptoms were established by such exposure and illness is there presumptive evidence that the later appearanec of chronic pulmonary disease was due to gas, as the chronic diseases encountered in soldiers who were gassed are indistinguishable pathologically, radiologically and clinically from those occurring in the civil community.

The hypothesis of "occult gassing" as a cause of later appearing pulmonary disease is completely negated by the studies of the effects of these substances on the bronchi. Pulmonary symptoms inevitably appear at the time when damage to the bronchial mucosa is sustained from gas.

The substances employed in chemical warfare are, as a chemical group, inhibitors of carcinogenesis by hydro-

carbons, and are not carcinogenic in themselves. They cannot, therefore, be impugned as direct ætiological factors in the later appearance of carcinoma of the bronchus.

When in any particular case it is considered that exposure to war gases led to the development of chronic bronchitis, emphysema or bronchiectasis, this conclusion can be based only on presumptive evidence in the form of an established train of symptoms originating at the time of an original severe pulmonary injury due to this cause. In the absence of such chronological evidence the factors operating in the civil community must be regarded as the causes of these conditions.

In view of the medical observations over many years subsequent to the use of chemical agents in warfare, it would appear that "the use of the asphyxiating shell is far less inhuman than the employment of submarine boats".

Reports of Cases.

CASE I.—A riveter, aged twenty-three years, enlisted on September 4, 1914. He was well nourished and had had no illnesses prior to his enlistment. He received a gunshot wound in the neck on September 28, 1915, but returned to duty with his unit on February 5, 1916. On April 9, 1918, he was gassed with mustard gas and was evacuated to the casualty clearing station and convalescent depot, and was unfit to return to his unit until June 9. During the time when he was evacuated it was observed that he suffered from inflammation of the eyes, hoarseness of voice and some tightness in the chest. On July 29, 1918, he again received mustard burns on the buttock, and whilst under treatment for these burns he again complained of "tightness in the chest". He was discharged from the convalescent depot on this occasion on September 5, 1918. He was discharged from the forces on February 15, 1919, but did not at that time claim any pension.

In 1948, when aged fifty-seven years, the soldier claimed a war pension and stated that ever since 1918 he had had a persistent cough and sputum. It was shown that he had not sought regular medical attention until 1941; but his employment record showed that he had had repeated absences from work because of bronchitis, and that the nature of his employment had been progressively lightened because of increasing dyspnœa on exertion.

On July 12, 1948, medical examination showed him to be in a state of very poor nutrition, and there were clinical and radiological signs of established chronic bronchitis and emphysema.

The soldier's workmates and the employer's records served to establish a clear chain of evidence of respiratory symptoms from the time of the gassing up to 1948, and in view of the severity of involvement in 1948 and the chronological sequence it was considered that presumptive evidence of association between gassing and chronic bronchitis was established.

There is some medical validity for this viewpoint, although such a conclusion cannot be regarded as scientifically established.

CASE II.—A labourer enlisted on January 22, 1916, when aged thirty-six years, and appeared healthy and well nourished. He proceeded overseas, and whilst in France was severely gassed at Passchendaele on October 12, 1917. He was evacuated to Le Tréport, with a cough, hoarseness of voice and pain in the chest. He was retained in hospital for twenty-seven days, and during this time his cough decreased. After transfer to a convalescent depot he felt fairly well, but noticed some shortness of breath on exertion and a persistent dry cough. He returned to his unit on December 9, after eight weeks' rest. On November 5, 1918, he developed influenza, which required treatment for fourteen days, and he was eventually discharged from the forces on August 12, 1919.

He returned to casual labouring work, but received treatment at intervals from 1920 onwards for "chronic bronchitis" associated with "fine crepitations at both lung bases". He made no claim for a war pension until 1930, when he was aged fifty-one years and had well-established clinical evidence of chronic bronchitis. His claim was rejected and he continued his work as a labourer, receiving intermittent treatment from his private practitioner until 1953, when at the age of seventy-three years he had well-developed emphysema of a bullous type, with chronic bronchitis. These conditions were eventually regarded as due to his war service and he received a war pension.

The presumptive evidence that exposure to gas caused this soldier's bronchitis and subsequent emphysema is scanty. Resumption of service in the field and the attack of influenza in 1918 negate the scientific validity of a claim that the bronchitis was due to gas exposure, whilst the age at the onset of symptoms is in accord with the clinical pattern observed amongst civilians. The evidence in this case of any permanent result from gas exposure is inadequate to indicate a clear association, but there is a possibility of such an association.

CASE III.—A clerk enlisted on December 6, 1916, when aged twenty-three years. He had had no illnesses prior to his enlistment and appeared well nourished. He served in France from October, 1917, and had no wounds or injury in the field. He proceeded to London on leave in August, 1918, and whilst there developed severe diarrhœa. He was treated in hospital for this condition from August 21 to October 10, 1918, and stated, many years later, that a medical officer at this time noticed a blister on his lip which resembled a "mustard burn". However, there was no history of undue exposure to gas, nor were there any respiratory symptoms noted or claimed to have existed in 1918. He was discharged from the forces as fit on August 12, 1919, and resumed his pre-war occupation.

In September, 1934, the soldier had an attack of influenza and was absent from work for two weeks, but had no other absences from work until he had a "chill" in 1940 and a further attack of influenza in 1944, when aged fifty-one years. In 1945, 1946, 1947 and 1948 he had repeated "colds" and occasional bronchitis, but satisfactorily performed his sedentary duties, although his recreational activities became progressively restricted. In May, 1949, he noticed severe restriction of his breathing, and this has become progressively more severe and associated with a persistent cough.

In February, 1953, there was clinical and radiological evidence of bullous emphysema with associated bronchitis. The soldier was then aged sixty years and his respiratory limitations were such as to prevent his employment.

A claim was made, in 1953, that the chronic bronchitis and emphysema were the late sequelæ of subclinical or "occult gassing" sustained in 1918. There is no doubt that the soldier was exposed to gas in 1918, as were all men serving in the front line in France; but it is quite clear that there were no respiratory effects or symptoms in 1918. The first suggestion of recurring respiratory symptoms arose in 1944, and the subsequent repeated respiratory infections and development of emphysema conforms in pattern to that which may be seen in any adult male in the sixth decade of life.

In this case there is neither scientific nor presumptive evidence to associate the chronic bronchitis and bullous emphysema, which became apparent over twenty years after service, with gas exposure during 1918.

Acknowledgements.

I wish to thank the Chairman of the Repatriation Commission for permission to use material available in the Repatriation Department and to publish this paper. The views expressed are my own and do not necessarily express the opinions of the Repatriation Department. Mr. C. B. Scott, representative in Australia of the British Ministry of Pensions, has been of considerable assistance in the collection of information, and the American Army Medical Service has also provided valuable data included in this study.

References.

BROWN, E. C. (1948), "Pulmonary Effects following Chronic Exposure to HS Vapor", "American Army Medical Division Reports", Army Medical Center, Maryland.

BUTLER, A. G. (1943), "Official History of the Australian Army Medical Corps, 1914-1918", Australian War Memorial, Canberra; Footnotes 6, 14, 12, 17, 31, 36, 47, 54; Tables 26, 811, 837.

CHRISTIE, C. V. (1951), "British Encyclopædia of Medical Practice", Second Edition, Butterworth, 5: 51.

COURTNEY, C. A. (1933), Confidential Report to Repatriation Commission.

MEAKINS, C. J. (1936), "The Practice of Medicine", Mosby, 146.

BRITISH WAR OFFICE (1941), "Medical Manual of Chemical Warfare", H.M. Stationery Office, 12: 37.

OSWALD, N. C., HAROLD, J. T., and MARTIN, W. J. (1953), "Clinical Pattern of Chronic Bronchitis", *Lancet*, 2: 638.

PRICE, G. B. (1938), "After Results of Gassing", *M. Press*, 196: 216.

ROBERTS, A. A. (1915), "The Poison War", Heinemann, London, Appendix VI: 142.

SERGENT, E. (1925), "The Respiratory Sequelæ of Poisoning by War Gases", *Presse méd.*, 33: 201.

STAEHELIN, R. (1920), "After Effects upon the Respiratory Organs of War Gas Poisoning", *Jahresk. f. ärtzl. Fortbild.*, 11: 17.

WARTHIN, A. S., and WELLER, C. V. (1919), "The Medical Aspects of Mustard Gas Poisoning", Kimpton, London.

WELLER, R. M. (1953), "Metaplasia of Bronchial Epithelium", *Am. J. Clin. Path.*, 3: 768.

WILLIS, R. A. (1948), "Pathology of Tumours", 1st Edition, Butterworth, 43.

WINTERNITZ, M. C. (1919), "Chronic Lesions of the Respiratory Tract", *J.A.M.A.*, 73: 689.

CORTICOTROPHIN AND CORTISONE IN IDIOPATHIC THROMBOCYTOPENIC PURPURA.

By H. N. ROBSON, M.B., Ch.B., F.R.C.P.E.,
Department of Medicine, University of Adelaide.

THE precise value of corticotrophin (ACTH) and cortisone in the management of idiopathic thrombocytopenic purpura has not yet been defined. Since the use of these substances in this condition was first reported (Robson and Duthie, 1950), experience appears to have been mixed, though on the whole favourable. (Leading article, 1952.) It is difficult to draw conclusions on personal experience because of the well-known tendency to spontaneous remission in this relatively uncommon disease. In presenting some further results with this form of treatment, it was therefore thought desirable and opportune to combine these results with other series reported in the literature to date. Some definite indication of the value, if any, of this form of therapy might then be obtained.

Ninety-six cases of idiopathic thrombocytopenic purpura have been collected from the literature—cases in which the diagnosis was based on the generally accepted criteria and in which ACTH or cortisone was given (Table I). To these may be added seven previously unreported cases which have been studied personally, and whose features are summarized in Table II. The total series of 103 cases has been classified into four main groups as shown in Table I.

Group I: Full Remission.

Group I includes only those cases in which hormonal therapy was followed by a complete and sustained clinical and hæmatological remission. Twenty-three cases fall into this group—approximately one-fifth of the total series, or about one-quarter of the total if cases in which splenectomy had previously been performed are set aside. The duration of the disease prior to hormonal treatment was stated in 18 cases, and in only five of these was it longer than a month. Otherwise, the cases in this group were not distinguishable from those in groups II or III.

Group II: Partial Remission.

Group II comprises those cases in which the authors have claimed a significant improvement on clinical or hæmatological grounds as a result of treatment. Included are three classes of patients: (i) those in whom bleeding ceased although hæmatological values were not fully restored to normal; (ii) those who responded to treatment, but in whom relapse occurred immediately on withdrawal of the hormones or while the treatment was still continuing; (iii) those who showed remission, but in whom splenectomy was performed before the extent and duration of such remission could be judged. Group II contains 34 cases, almost one-third of the total series. The previous duration of the disease was stated in 17 instances, and in 11 it was longer than a month. In 19 cases the response to ACTH or cortisone may be termed "vascular" only—that is, remission of bleeding, with improvement in the results of capillary tests and in bleeding time, but without a significant platelet increase—while in the remaining 15, platelet counts reached normal or supernormal figures at some point during the hormonal treatment. Splenectomy was performed after or during the administration of hormone in 24 of these cases, with immediately satisfactory results.

Group III: No Response.

Cases were allocated to this group on the author's verdict that no significant response had occurred. Some workers chose the platelet level as the main measure of response, and the result of treatment was recorded as negative if there was no significant elevation of platelet counts, although in some cases reference is made to "cessation" or "diminution of purpura" (Harrington *et alii*, 1953). Such cases are included here, although they seem similar to those in Group II, in which the claim of a "vascular" response was made (Faloon *et alii*, 1952; Stefanini *et alii*, 1952).

Twenty-seven cases were included in this group. The duration of the disease, stated in 16 cases, was longer than a month in eight. Subsequent splenectomy was recorded as being effective in 16 cases and ineffective in seven. It is noteworthy that in many of these cases in which the hormones had failed to evoke any response, the result of subsequent splenectomy was described as "complete" or "excellent" (Myers *et alii*, 1952; Harrington *et alii*, 1953; Adamson *et alii*, 1953).

Group IV: Previous Splenectomy.

All patients in Group IV were given the hormones because of recurrence of the disease after splenectomy had been performed at varying intervals previously. There were 19 cases in this category. Treatment produced results in 14 cases, the response being described as "full" in seven instances and "partial" in the remainder. Remissions, even when complete, tended to be of short duration. On the other hand, such remissions were reproduced several times in the same individual (Evans and Liu, 1951; Jacobson and Sohler, 1952; Harrington *et alii*, 1953). In five instances there was no response to the hormonal therapy.

Methods of Treatment.

In the cases of Group I in which full remission was obtained, and in which the mode of treatment was detailed, ACTH was used on 11 patients and cortisone on nine. In Group II, partial remission was due to ACTH on nine occasions and to cortisone on 12. In those cases in which both drugs were used, there is some indication that cortisone given by mouth was the more effective (Faloon *et alii*, 1952; Adamson *et alii*, 1953). In Group III, ACTH was recorded as having failed on 12 occasions and cortisone on 14. Both drugs were used without effect on nine patients. In cases in which relapse occurred after splenectomy (Group IV), ACTH was used to produce remission in seven and cortisone in five. In one case (Davidson *et alii*, 1952) ACTH failed while cortisone caused a transient remission. There was no indication in any of these reports that there was any advantage in increasing the dosage of ACTH above 150 milligrammes daily or that of cortisone above 300 milligrammes daily.

Discussion.

There are three main points about which this survey may provide information. Is the administration of ACTH or cortisone of any value in this disease? If so, what are the indications for their use? Do the results give any information as to the action of these substances?

Value of Hormonal Therapy.

Some response was obtained by hormonal treatment in 71 of the total series of 103 cases. It may be that this is not a true measure of over-all experience, since positive results are perhaps more likely to have been reported. On the other hand, the proportions of success and failure are roughly similar in the larger recorded series (Table I). The cases in Group I, in which a full remission followed hormonal treatment, do not help much in this assessment. More than two-thirds of this group of patients had shown manifestations of the disease for less than a month, and

TABLE I.

Classification of Reported Cases of Idiopathic Thrombocytopenic Purpura Treated with ACTH or Cortisone.

Authors.	Group I: Full Remission.	Group II: Partial Remission.	Group III: No Response.			Group IV: Relapsed after Splenectomy.	
			No Further Treatment Recorded.	Splenectomy Later.			
				Effective.	Not Effective.	Good Response.	No Response.
Robson and Duthie, 1950	1	1	—	—	—	—	—
Evans and Liu, 1951	—	—	—	—	—	1	—
Hyman, 1951	2	—	—	—	—	—	—
Ragan, 1951	—	—	—	—	—	2	—
Wightman, 1951	—	2	—	—	—	—	—
Wilson and Eisemann, 1951	4	2	—	6	—	1	—
Wintrobe *et alii*, 1951	—	1	—	—	—	—	—
Davidson *et alii*, 1952[1]	3	1	1	2	—	1	1
Faloon *et alii*, 1952	1	2	—	—	—	1	—
Jacobson and Sohler, 1952	—	1	—	—	—	2	—
Medical Research Council Report, 1952[1]	1	4	3	—	—	—	2
Meyers *et alii*, 1952	5	10	—	1	—	1	—
Stefanini *et alii*, 1952	1	4	—	—	—	3	—
Harrington *et alii*, 1952	2	3	—	4	6	2	2
Adamson *et alii*, 1953	1	1	—	1	—	—	
Present series	2	2	—	2	1	—	—
Totals	23	34	4	16	7	14	5

[1] There may be duplication here, as an unspecified number of cases included in the Medical Research Council report (1952) were later reported by Davidson *et alii* (1952).

as Hirsch and Dameshek (1951) have emphasized, about one in every five patients with idiopathic thrombocytopenic purpura examined in the first attack will show a spontaneous, long-lasting remission. However, at least five patients with previous histories of longer than a month are recorded as having responded fully. Nevertheless, it is clear that many of the apparent remissions in this group could have been spontaneous.

The cases in Group II, in which partial remission occurred, are more informative. Recorded information shows that six of these cases were "acute" and 11 were "chronic". Spontaneous remission must also be considered in this group, though the arm of coincidence would have to be stretched further. However, in this group are included many cases in which there was a full or partial remission while the hormone was being administered, with an immediate relapse on withdrawal of the drug (Wintrobe *et alii*, 1951; Faloon *et alii*, 1952; Wilson and Eisemann, 1952; Stephanini *et alii*, 1952). In other cases such remissions were reproduced in the same patient on several occasions (Robson and Duthie, 1950; Davidson *et alii*, 1952; Adamson *et alii*, 1953). Such examples strongly indicate a definite action of ACTH and cortisone in this condition, and also suggest that the results in Group I may not have been altogether coincidental.

The negative results in Group III are an obvious measure of the limitations of this form of treatment. One most interesting feature which emerges from a study of these cases is the fact that failure to respond to hormonal therapy does not apparently preclude a subsequent good result from splenectomy.

TABLE II.

Details of Seven Cases of Idiopathic Thrombocytopenic Purpura Treated with ACTH or Cortisone.

Case Number.	Patient's Sex and Age. (Years.)	Duration of Disease.	Treatment.	Result.
I	F., 32	3 weeks.	ACTH, 100 milligrammes daily for 10 days.	Capillary resistance and bleeding time normal on second day. Bleeding stopped. Platelets normal on sixth day. Full remission continued to one year.
II	F., 21	7 days.	ACTH, 100 milligrammes daily for 5 days.	Capillary resistance and bleeding time normal on second day. Bleeding stopped. Platelets normal on eighth day. Full remission continued to seven months.
III	F., 53	3 years.	ACTH, 100 milligrammes first day, 75 milligrammes daily for 6 days.	Capillary resistance and bleeding time normal on third day. Bleeding stopped. Platelets subnormal rise, splenectomy on seventh day with good result.
IV	M., 27	6 months.	(i) ACTH, 100 milligrammes daily for 7 days. (ii) Cortisone, 200 milligrammes first day, 100 milligrammes daily for 10 days.	(i) Capillary resistance rise, bleeding time fall, no change in platelets, bleeding reduced. (ii) Further improvement. Capillary resistance and bleeding time subnormal, platelet rise. Splenectomy on eighteenth day—good result.
V	M., 6	2 weeks.	(i) ACTH, 75 milligrammes first day, 50 milligrammes daily for 5 days. (ii) Cortisone, 100 milligrammes daily for 10 days.	(i) Capillary resistance rise, bleeding time, platelets, no change. Bleeding continued. (ii) No further improvement. Splenectomy on twenty-first day—excellent result.
VI	F., 5	4 weeks.	ACTH, 75 milligrammes first day, 50 milligrammes daily for 12 days.	Capillary resistance rise. Transient improvement, bleeding time and platelets. Bleeding recurred on eighth day. Splenectomy on thirteenth day—excellent result.
VII	M., 18	5 weeks.	Cortisone, 200 milligrammes first day, 100 milligrammes daily for 8 days, 300 milligrammes daily for 2 days.	Capillary resistance rise, suboptimal. No platelet change. Bleeding continued. Splenectomy on fourteenth day—poor result.

The cases in Group IV are perhaps the most informative of the whole series. Two-thirds of these patients, all of whom had relapsed after splenectomy, responded to ACTH or to cortisone. The coincidence of spontaneous remission is least likely in this group and, as has been mentioned, the results of hormonal therapy were reproducible in several instances.

The results of this survey indicate reasonably clearly that ACTH or cortisone has a definite effect, with limitations, on the manifestations of this disease and is of value in management.

Indications for Hormonal Therapy.

There is no evidence, so far available, that ACTH or cortisone has any adverse effect in this condition. In approximately two out of every three patients who have not been subjected to splenectomy, hormonal therapy may control or diminish the hæmorrhagic manifestations. Even if the response is only partial, the risks of bleeding are diminished, and natural remission is awaited or splenectomy undertaken with less sense of urgency. If no response is obtained within ten days, there is little point in continuing with this treatment. Splenectomy may then be undertaken with apparently undiminished chances of success. Hormonal therapy would seem to be well worthy of trial in the treatment of any patient who has relapsed after splenectomy.

Action of Cortisone in Idiopathic Thrombocytopenic Purpura.

The ætiology of idiopathic thrombocytopenic purpura remains obscure, though there is accumulating evidence that a number of different mechanisms may be involved in the production of this syndrome (Harrington *et alii*, 1953; Stefanini *et alii*, 1953). The present survey gives little definite indication of the possible mode of action of ACTH or cortisone except that the findings are entirely compatible with the suggestion that their action is predominately on the vascular component of the bleeding tendency.

Purpuric bleeding is related both to vascular fragility and to thrombocytopenia, which are themselves interrelated; but bleeding is not constantly produced by either of these factors alone. It seems probable that in idiopathic thrombocytopenic purpura both factors operate, but that the relative importance varies from one case to another. ACTH and cortisone improve capillary resistance in normal and in pathological states (Robson and Duthie, 1951, 1952), and it was this observation which prompted the use of these substances in idiopathic thrombocytopenic purpura. A primarily vascular action of cortisone would explain the events seen in so many of these cases during hormonal therapy—namely, improvement in the results of vascular tests and diminution in bleeding without or preceding any significant increase in the number of platelets. Splenectomy probably both affects the vascular defect (Robson, 1949), and in successful cases has an additional direct effect on the mechanism causing thrombocytopenia. This concept would explain the variability of the action of cortisone, the transitory nature of the results in many cases, the success of splenectomy when hormone therapy has failed, and the success of hormonal therapy when splenectomy has failed.

Summary.

An attempt has been made to assess the value of ACTH or cortisone in the treatment of idiopathic thrombocytopenic purpura on the basis of a survey of 96 examples reported in the literature and a further seven personally studied cases. Beneficial effect of varying degree was claimed in 71 of these cases. The method of administration, indications for use and possible mode of action of these hormones are discussed briefly. It is concluded that this treatment is of value in the management of this disorder.

References.

Adamson, D. G., Walker, W., and Macintosh, A. E. (1953), "ACTH and Cortisone in Idiopathic Thrombocytopenic Purpura and Schönlein-Henoch Purpura", *Brit. M. J.*, 2: 656.

Davidson, L. S. P., Girdwood, R. H., and Swan, H. T. (1952), "Effect of ACTH and Cortisone Therapy in Blood Disorders", *Brit. M. J.*, 2: 1059.

Evans, R. S., and Liu, C. K. (1951), "Effect of Corticotrophin on Chronic, Severe, Primary Purpura", *Arch. Int. Med.*, 88: 503.

Falcon, W. W., Greene, R. W., and Lozner, E. L. (1952), "The Hemostatic Defect in Thrombocytopenia as Studied by the Use of ACTH and Cortisone", *Am. J. Med.*, 13: 12.

Harrington, W. J., Sprague, C. C., Minnich, V., Moore, C. V., Ahlvin, R. C., and Dubach, R. (1953), "Immunologic Mechanisms in Idiopathic and Neonatal Thrombocytopenic Purpura", *Ann. Int. Med.*, 38: 433.

Hirsch, E. O., and Dameshek, W. (1951), "Idiopathic Thrombocytopenia", *Arch. Int. Med.*, 88: 701.

Hyman, G. A. (1951), "Idiopathic Thrombocytopenic Purpura", *Am. J. Med.*, 11: 499.

Jacobson, B. M., and Sohier, W. D. (1952), "The Effects of ACTH and Cortisone on the Platelets in Idiopathic Thrombocytopenic Purpura", *New England J. Med.*, 246: 247.

Leading Article (1952), "ACTH and Cortisone in Hypersplenic Syndromes", *J.A.M.A.*, 149: 484.

Medical Research Council Report (1952), "The Treatment of Blood Disorders by ACTH and Cortisone", *Brit. M. J.*, 1: 1261.

Meyers, M. C., Miller, S., Linman, J. W., and Bethell, F. H. (1952), "The Use of ACTH and Cortisone in Idiopathic Thrombocytopenic Purpura and Acquired Hæmolytic Anæmia", *An. Int. Med.*, 37: 352.

Ragan, C. (1951), "Proceedings of the Second Clinical ACTH Conference", edited by J. R. Mote, Churchill, London, 2: 178.

Robson, H. N. (1949), "Idiopathic Thrombocytopenic Purpura", *Quart. J. Med.*, 18: 279.

Robson, H. N., and Duthie, J. J. R. (1950), "Capillary Resistance and Adrenocortical Activity", *Brit. M. J.*, 2: 971.

——— (1952), "Further Observations on Capillary Resistance and Adrenocortical Activity", *Brit. M. J.*, 1: 994.

Stefanini, M., Santiago, E. P., Chatterjea, J. B., and Dameshek, W. (1952), "Corticotrophin and Cortisone in Idiopathic Thrombocytopenic Purpura", *J.A.M.A.*, 149: 647.

Stefanini, M., Dameshek, W., Chatterjea, J. B., Adelson, E., and Mednicoff, I. B. (1953), "Studies on Platelets, IX", *Blood*, 8: 26.

Wightman, K. J. R. (1951), "Proceedings of the Second Clinical ACTH Conference", edited by J. R. Mote, Churchill, London, 2: 179.

Wilson, S. J., and Eisemann, G. (1952), "The Effect of ACTH and Cortisone on Idiopathic Thrombocytopenic Purpura", *Am. J. Med.*, 13: 21.

Wintrobe, M. M., Cartwright, G. E., Palmer, J. G., Kuhns, W. J., and Samuels, L. T. (1951), "Effects of Corticotrophin and Cortisone on the Blood in Various Disorders in Man", *Arch. Int. Med.*, 88: 310.

HYPERTROPHIED HEARTS NOT DUE TO VALVULAR OR RENAL DISEASE: II.

By J. B. Cleland, C.B.E., M.D.,

Emeritus Professor of Pathology, University of Adelaide; Visiting Specialist, Repatriation General Hospital, Adelaide.

In a previous paper (Cleland, 1954) stress was laid on the occasional occurrence of cardiac hypertrophy and subsequent failure without any valvular or renal cause, and in the absence of hypertension. It was also pointed out that in the seventh thousand of our tabulated post-mortem examinations at the Royal Adelaide Hospital no less than 14·9% of subjects had hearts which were heavier than normal without a valvular or renal cause for the enlargement. The present paper deals with such hypertrophied hearts as found in 6000 of these post-mortem examinations between 1925 and 1948. (See *The Medical and Scientific Archives of the (Royal) Adelaide Hospital.*)

Here it may be pertinent to ask what range of weight of the heart may be found in healthy grown-up men and women in Australia. Until the advent of the motor-car, adequate data along these lines would have taken a considerable time to accumulate. However, fatal motor-car accidents are now so common that the opportunity should be seized by those in a position to do so to obtain the needed information. Means, usually lacking, should be available in the post-mortem room for weighing the body as a whole. The length and breadth and general appearance (small, large, thin, fat) of the body are usually noted. I presume that a heavy burden of subcutaneous and abdominal fat, developing as years go by, does not cause any appreciable increase of work for the heart, though besides the fat there must be an increase in blood vessels and supporting stroma, and also an increase in superficies.

Normal ranges or averages, based probably on European data, vary. Thus I have for males 270 to 360 grammes (9·5 to 12·75 ounces) and 390 grammes (13·75 ounces), and for females 250 to 280 grammes (8·75 to 10 ounces) and 340 grammes (12 ounces). I have usually recorded my weights in ounces and have come to consider that a weight of about 11 to 13 ounces (310 to 370 grammes) represents about the normal in South Australia. In 1946 and 1947 I kept a note of the recorded weights of the hearts of 74 men and 39 women. The heart weighed 14 ounces or under in 35 of the men—nearly half—and in 27 of the women—nearly three-quarters. In two women it weighed seven ounces; in two men and nine women, nine ounces; in one woman, nine and a half ounces; in ten men and four women, eleven ounces; in six men and six women, twelve ounces; in four men and four women, fourteen ounces. I have usually considered 15 ounces (425 grammes) as showing hypertrophy. Thirty-seven of the men (half of them), on this scale, had hypertrophied hearts, but only about one-quarter of the women. These are hospital patients suffering from diseases, some of which may reduce the weight of the heart, as in emaciation, and others increase it. Incidentally I may mention that in several post-mortem examinations on accident subjects I have been struck by the large size of the liver in some apparently healthy persons. I usually consider that in hospital patients the weight of the liver is about 50 ounces (1418 grammes) and I find the range for normal has been given as 51 to 58 ounces (1440 to 1680 grammes); but some of these livers weighed 70 ounces (about 2000 grammes) or more. For example, a young man in his twenties had been murdered by being shot through the lung; he was five feet ten inches in height, and his liver weighed 70 ounces (1988 grammes) and his heart 14 ounces (400 grammes). A man, aged twenty-eight years, had died from hanging; his height was six feet five inches, and his liver weighed 76 ounces (2158 grammes) and his heart 12·5 ounces (356 grammes). A man, an alcoholic, had been drowned; his liver weighed 69 ounces. The necessity to collect these data is obvious.

In these 6000 post-mortems, 243 subjects had hypertrophied and dilated hearts and had in most instances died from congestive cardiac failure. These figures suggest that in South Australia about 4% at least of adults who die, die from such a cause. Such a common cause of death should be reflected in the official statistics, which are based on the International List of the Causes of Death. In the "Statistical Register of the State of South Australia for the Year 1950 to 1951, Part II", diseases of the circulatory system are shown as contributing 2394 deaths. Of these, 87 are classified as chronic rheumatism (mostly of course being due to valvular disease), 982 as "arterio-sclerotic heart disease, including coronary disease" (Item 420), and 744 under "other myocardial degenerations", 211 under hypertension and 99 under general arteriosclerosis, and 271 under other headings. In other words, 1726 deaths are by inference due to coronary disease, but only about 310 are attributed to the cardiac effects of hypertension. As our records at the Royal Adelaide Hospital show that about as many people die from failure of an hypertrophied heart as from coronary disease, something appears to be radically wrong with the signing of many of these death certificates.

The term "arteriosclerotic heart disease" used by doctors in signing the death certificate, and endorsed in the "Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death", Sixth Revision (1948), Volume I, and so necessarily accepted by the statistician, is most unfortunate. Its including "coronary disease" implies that it refers to the coronary vessels and really means "athero-sclerosis", which I prefer to call atheroma (when there is thus no confusion with arteriosclerosis). But does the medical man using the term mean degeneration of the coronaries or does he really mean, in most cases, cardiac failure from hypertension—a very different thing, though the two are often combined? For clarity, Item 420 should be "coronary disease" alone; then the medical man and the statistician would be forced to place the appropriate instances under "hypertensive disease" (Items 440 to 447). Also Item 450 ("general arteriosclerosis") has little meaning as a cause of death other than in hypertension, and should be included under "hypertensive disease" and not under "diseases of arteries" with such killing conditions as aneurysm.

It seems reasonable to attribute most of these 243 hypertrophied hearts to essential hypertension, though in many of the cases the blood pressure may not have been taken or may have fallen before death. As shown by their weights, these hearts are hypertrophied and the hypertrophy may be in some cases very great; the heaviest heart under this category (that of a woman, aged fifty years) weighed 37 ounces. Hypertrophy must indicate that the heart has had more work than normal to do. The only exceptions to this explanation of the hypertrophy would be in the case of splanchnomegaly, which is characteristic of acromegaly. Here all the organs are enlarged—the liver, kidneys, and so on, the heart included. In fact, one of our largest hearts was found in a case of acromegaly; it weighed 42 ounces, and part of the hypertrophy at least must be attributed to the general enlargement of organs. However, we can hardly assume that many, if any, of these other hypertrophied hearts owe their enlargement to a pituitary cause. In the absence of valvular or renal diseases we are forced to consider that the increased work the hearts have had to do has in most cases been against an increase of pressure in the blood-stream.

In this paper it is not proposed to consider what causes may give rise to this increase in pressure, and whether the thickening of small arterioles, which may accompany the condition, is primary or secondary. I think we are justified in considering that the hypertrophy is usually due to hypertension, and, as the cardiac condition is the most important feature in these cases, and is as a matter of fact the cause of death, the cases should be classified under diseases of the heart itself, with some such caption as cardiac hypertrophy and failure from hypertension or hyperpiesis.

In the first thousand post-mortem examinations tabulated the importance of this condition was not recognized. In those days between 1920 and 1925 one tried to place these cases under the category of hypertrophied hearts secondary to renal fibrosis, even though the renal disease often seemed insignificant. Consequently in this review of our cases I have excluded the first thousand and I think the particulars for the second thousand may be an underestimate.

In addition to the cases in which the heart had actually failed were 5% in which the heart was enlarged but showed as yet no signs of failure, as well as about 2·4% in which the hypertrophied heart and high blood pressure were associated with cerebral hæmorrhage, and 1·8% in which they were associated with infarction of the heart. Thus in all about 13% of adults who die in the Royal Adelaide Hospital have hypertrophied hearts, probably owing to hypertension.

The ages at which the patients have died from failure of the hypertrophied heart are presented in Table I, the results in the last 6000 post-mortem examinations being given.

The youngest subject was a lad, aged sixteen years, whose blood pressure had been 185 millimetres of mercury, systolic, and 110 millimetres, diastolic, and whose heart weighed 25 ounces (710 grammes). The next was a man, aged twenty-two years, whose heart weighed 18·5 ounces (525 grammes); then came a man, aged twenty-three years, whose heart weight was not given; next a man, aged twenty-seven years, whose heart was only slightly hypertrophied, weighing 17 ounces; and then a man, aged twenty-nine years, with a heart weighing 20 ounces (568 grammes).

The two youngest women were both aged thirty-four years, one having a heart which weighed 22·5 ounces (639 grammes) and the other a heart weighing 20 ounces (568 grammes).

The largest hearts in the last 5000 post-mortems in these cases in male subjects were as follows: age thirty-eight years, 34 ounces (965 grammes); age forty-four years, 34 ounces (965 grammes); age thirty-seven years, 33 ounces (937 grammes); age seventy-one years, 32·5 ounces (922

TABLE I.

Failure of the Hypertrophied Heart.

Age Group. (Years.)	Males.							Females.							Both Sexes.
	Second Thousand.	Third Thousand.	Fourth Thousand.	Fifth Thousand.	Sixth Thousand.	Seventh Thousand.	Total.	Second Thousand.	Third Thousand.	Fourth Thousand.	Fifth Thousand.	Sixth Thousand.	Seventh Thousand.	Total.	
Teens ..	—	—	1	—	—	—	1	—	—	—	—	—	—	—	1
20's ..	—	—	1	1 (27)	2	—	4	—	—	—	—	—	—	—	4
30's ..	1	2	4	—	1	3	11	2	—	—	—	2	—	4	15
40's ..	3	2	2	4	7	2	20	—	3	—	3	2	3	11	31
50's ..	3	6	10	6	7	11	43	2	2	3	4	2	7	20	63
60's ..	7	5	11	12	5	9	49	2	2	2	2	—	—	10	59
70's ..	6	5	7	15	8	6	47	1	1	1	1	—	—	4	51
80's ..	1	3	1	5	3	3	16	—	1	—	2	—	—	3	19
90's ..	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	21	23	37	43	33	34	191	7	9	6	12	8	10	52	243

grammes); age sixty-eight years, 32 ounces (908 grammes); age sixty-four years, 31·5 ounces (894 grammes); age fifty-four years, 31 ounces (880 grammes); age fifty-three years, 30·75 ounces (873 grammes); ages thirty-six, thirty-eight and seventy-two years, 30 ounces (851 grammes); ages fifty-nine and sixty-six years, 29·75 ounces (844 grammes); ages thirty-four and fifty-nine years, 29·5 ounces; age sixty-five years, 29 ounces (823 grammes); 11 subjects, 30 ounces (851 grammes) or over.

In female subjects, the largest hearts were distributed as follows: age 50 years, 37 ounces (1050 grammes); age fifty-two years, 31 ounces (880 grammes); age sixty-eight years, 30 ounces (851 grammes); age fifty-one years, 26 ounces; age forty-nine years, 28 ounces (795 grammes); three subjects over 30 ounces (851 grammes).

Among male subjects, the numbers of hearts which weighed 20 ounces or over were as follows: in the third thousand, 16 (out of 23); in the fourth thousand, 21 (out of 37); in the fifth thousand, 26 (out of 42); in the sixth thousand, 17 (out of 33); in the seventh thousand, 25 (out of 34); a total of 105 out of 170.

Among female subjects, the numbers of hearts which weighed 20 ounces or over were as follows: in the third thousand, four (out of nine); in the fourth thousand, two (out of six); in the fifth thousand, five (out of twelve); in the sixth thousand, five (out of eight); in the seventh thousand, five (out of ten); a total of 21 out of 45.

The grand total of hearts which weighed 20 ounces or over was 126 out of 215.

From the foregoing it will be seen that these hypertrophied hearts are commoner in men than in women, even when allowance is made for the fact that about two post-mortem examinations are made on men for one on a woman.

Whatever the cause of this hypertrophy is, it is something operating more frequently in males than in females, though the size of the heart may be even greater sometimes in the latter.

The chief incidence of the disease is in the sixties, which are closely followed by the seventies and then by the fifties. There was only one patient aged under twenty years; five patients were in their twenties, and 14 in their thirties. With the exception of children, no age seems exempt.

Hypertrophied Hearts and Cardiac Infarction.

The heart was hypertrophied without adequate valvular or renal cause—that is, probably from essential hypertension—in 108 out of 189 cases of infarction in the last six thousand tabulated post-mortem examinations (ending with the seventh thousand) at the Royal Adelaide Hospital. This constitutes 1·8% of all post-mortem examinations. One heart was weighed with a very large ante-mortem clot in the ventricle, so that much of the 45 ounces (1277 grammes) was probably attributable to this clot. The weights of some of the others were as follows in descending series: 35·5 ounces (1008 grammes); 30 ounces (851 grammes); 29·75 ounces (844 grammes); 28 ounces (795 grammes); 27 ounces (766 grammes); 26 ounces (738 grammes); 25 ounces (710 grammes) (with ante-mortem clot in cardiac chamber); 24 ounces (681 grammes) (four cases); 23 ounces (653 grammes) (four cases); 22 ounces (624 grammes) (four cases); 21·5 ounces (610 grammes); 21 ounces (596 grammes) (3 cases); and 20 ounces (568 grammes) (7 cases).

It will be seen that three hearts weighed 30 ounces (851 grammes) or more, eight weighed 25 ounces (710 grammes) or more, and 30 weighed 25 ounces (568 grammes) or more. As the weights of some of these hypertrophied hearts were

TABLE II.

Cerebral, Cerebellar or Pontine Hæmorrhage with Hypertrophied Hearts.

Age Group. (Years.)	Males.					Females.					Both Sexes.
	Fourth Thousand.	Fifth Thousand.	Sixth Thousand.	Seventh Thousand.	Total.	Fourth Thousand.	Fifth Thousand.	Sixth Thousand.	Seventh Thousand.	Total.	
Teens	—	—	—	—	—	—	—	—	—	—	—
20's	—	—	—	—	—	—	—	1 (23)	—	1	1
30's	—	2	—	—	2	—	2	1 (36)	—	3	5
40's	2	3	2	1	8	5	4	3	1	13	21
50's	4	9	3	8	24	1	2	7	5	15	39
60's	4	1	3	8	16	2	1	2	—	5	21
70's	1	1	2	1	5	1	2	—	—	3	8
80's	—	—	—	—	—	1	—	—	—	1	1
90's	—	—	—	—	—	—	—	—	—	—	—
Total	11	16	10	18	55	10	11	14	6	41	96

not recorded, probably nearly one-third of these 108 hypertrophied hearts weighed 20 ounces or more.

Cardiac Hypertrophy Only without Obvious Failure.

In the last four thousand autopsies there were respectively 48, 64, 44 and 37 (total 189) instances in which the heart was hypertrophied without adequate valvular or renal cause, but the patient had not died from its failure but incidentally from other causes. This adds another 5% to the incidence of cardiac hypertrophy without adequate renal or valvular cause in these Royal Adelaide Hospital cases. However, only 25 of the 189 subjects had hearts weighing more than 20 ounces (568 grammes), so that the hypertrophy in most cases was only slight. Some of these 25 cases are worth mentioning briefly, as follows. A male subject, aged sixty-five years, had died of a ruptured appendix; he had a heart weighing 30·5 ounces (866 grammes). A male subject, aged fifty-nine years, had died of diffuse cellulitis of the face; the heart weighed 29·75 ounces (844 grammes). A male subject, aged sixty-nine years, had died of hæmorrhage from a duodenal ulcer; the heart weighed 29 ounces (823 grammes). A male subject, aged forty-five years, had died of diverticulitis and peritonitis; the heart weighed 27 ounces (766 grammes). A male subject, aged seventy-eight years, had died of intestinal obstruction from a gall-stone; the heart weighed 24 ounces (681 grammes). A male subject, aged fifty-two years, had died of uræmia from ascending pyelonephritis; his heart weighed 23·5 ounces (667 grammes). A female subject, aged fifty-five years, had died from intestinal obstruction; her heart weighed 22 ounces (624 grammes). A female subject, aged sixty-five years, had died of a gastric ulcer; her heart weighed 22 ounces (624 grammes). A male subject, aged seventy-three years, had died of carcinoma of the rectum; his heart weighed 21 ounces (596 grammes). A male subject, aged sixty-three years, had died of a foul empyema; his heart weighed 20·75 ounces (589 grammes). A male subject, aged seventy-four years, had died of lobar pneumonia; his heart weighed 20·5 ounces (582 grammes). A male subject, aged fifty-four years, had died of a staphylococcal empyema; his heart weighed 20 ounces (568 grammes).

Hypertrophied Hearts with Cerebral Hæmorrhage.

In the last 4000 autopsies there were 96 examples of hæmorrhage into the brain (cerebral, cerebellar or pontine) with hypertrophy of the heart not due to valvular or obvious renal conditions—that is, about 2·4% of deaths in hospital cases (see Table II). Over the same period there were 94 examples, 54 of them in women, of cerebral hæmorrhages in which the heart was not recorded as hypertrophied; but in several of these cases permission had been granted only to examine the head. There were further cases of cerebral hæmorrhage associated with renal fibrosis which are not dealt with here.

It thus appears on these statistics that about 2·4% of hospital patients die from hæmorrhage into the brain and have hypertrophied hearts, suggesting that they had suffered from hyperpiesis, and that another 2·4% die from such hæmorrhages, but there is no evidence in the record that the heart was hypertrophied from essential hypertension or from chronic nephritis and renal fibrosis.

It will be noticed that the ages of these subjects of cerebral hæmorrhage tend to be lower than those associated with failure of the hypertrophied heart. The chief incidence is in the fifties in males and then the sixties, and in the forties and fifties in women. Failure of the hypertrophied heart occurs chiefly in the sixties and the seventies in males and in the fifties and sixties in women.

Reference.

CLELAND, J. B. (1954), "Hypertrophied Hearts not Due to Valvular or Renal Disease: I", M. J. AUSTRALIA, 1: 432.

Reports of Cases.

TWO UNUSUAL COMPLICATIONS OF MECKEL'S DIVERTICULUM.

By H. DONOUGH O'BRIEN,
Surgeon Superintendent, Public Hospital, Burnie, Tasmania.

SOME degree of Meckel's diverticulum occurs in from 1·5% to 2% of persons, and in about 25% of cases heterotopic tissue is present in the diverticulum. Although sometimes inoffensive, this vestige of the primitive yolk sac often produces trouble in the abdomen, and must always be borne in mind as a possible source of mischief. Probably the commonest complication caused by the diverticulum is intestinal hæmorrhage, but perforation with peritonitis, intussusception, band obstruction, acute inflammation, knots, umbilical fistula and cysts have been frequently reported. It is not proposed to make any survey of the literature, however, but only to report two rather complex cases recently experienced.

Case I.

In October, 1951, a male cycle shop attendant, aged sixteen years, was admitted to the Burnie Public Hospital, just after midnight, complaining of lower abdominal pain present since the previous morning. He had had no nausea or vomiting, the bowels had moved normally, and there had been no significant urinary disturbance. He had had no previous attacks of this nature. The boy had suffered from a number of minor congenital abnormalities including left syndactyly, which had been successfully treated at another hospital. He had had an operation for hernia in childhood, and five years prior to his admission to hospital he had been treated in another hospital for "abscess on the spine"; but the exact nature of this complaint was not determined. His general condition had been good.

Examination showed him to be a pale youth, in some pain, with *fætor oris* but a fairly clean tongue; abdominal movements were restricted, and tenderness and guarding were present in the right iliac fossa; the temperature was 99·4° F., and the pulse rate 120 per minute; examination of the urine revealed no abnormality. This seemed to be a fairly typical case of acute appendicitis, and immediate operation was carried out under "open ether" anæsthesia. The abdomen was opened through a McBurney incision, and some blood-stained free fluid was found in the peritoneal cavity. The appendix was covered by a film of fibrin, and when this was wiped off, the organ appeared fairly normal, but was nevertheless removed. The terminal portion of the ileum was now inspected and noted to contain some dark fluid material, but the significance of this was not appreciated until later. The mesentery contained some grossly enlarged lymphatic glands, some of which appeared to be calcified, but there was no sign of recent acute inflammation. About two feet from the ileo-caecal valve, the ileum was acutely kinked by an antemesenteric veil, apparently of congenital origin. This might have caused some intestinal obstruction, but there was no evidence that it had caused a degree of strangulation sufficient to account for the blood-stained free fluid in the peritoneal cavity. The veil was divided and the bare areas were peritonealized. The gut was then followed up further to approximately the level of the lower part of the jejunum, when it could no longer be delivered. Exploration with the finger now revealed a small mass adherent to the anterior abdominal wall about the back of the umbilicus. This was freed by blunt dissection, and delivered, a mass about the size of a golf ball being revealed, attached to the antemesenteric border of the jejunum. The mass was covered with fibrin, and near its junction with the jejunum there was an area of induration about a perforation one-eighth of an inch in diameter, from which some blood-

stained fluid escaped. The appearance was very like that of a perforated peptic ulcer of the pyloric region, and a diagnosis of bleeding and perforated peptic ulcer of ectopic tissue in a Meckel's diverticulum was made. The offending segment of jejunum was resected, continuity of the bowel was restored by end-to-end anastomosis, and the abdomen was closed without drainage. Convalescence was uneventful, and the patient was discharged home on the tenth post-operative day. He was followed up in the out-patient department for two years, during which time there seemed to be no trouble attributable to his condition at operation.

The portion of resected gut including the diverticulum was sent to the Launceston General Hospital for preparation and examination of sections, but unfortunately was mislaid, so that it was rather dried and distorted by the time it reached the pathologist, Dr. M. P. K. Shoobridge. However, he was able to reconstruct the pathological condition as one of intussusception of a Meckel's diverticulum with perforation in an area of chronic inflammatory tissue situated at the junction of the diverticulum with the jejunum. He could find no evidence of oxyntic cells, but was able to report the presence of Brunner's glands, which in his opinion was to be taken as evidence of the existence of ectopic tissue. The final diagnosis therefore was perforation and hæmorrhage from an area of chronic inflammation, possibly a chronic peptic ulcer, in a Meckel's diverticulum which contained ectopic tissue, and was also the site of an intussusception.

Case II.

The second case had a less fortunate outcome. A farmer, aged fifty years, was admitted to the Burnie Public Hospital at midday on March 24, 1952, complaining of abdominal pain and vomiting. He had always been well until two days previously, when he went to a race meeting and missed his dinner, having a hurried meal of two meat pies and some ice-cream instead. At midnight that night he had been awakened with pain in the left iliac fossa, but when he was examined by his own doctor about midday the following day he had improved somewhat. However, he relapsed later, and was referred to hospital, where he was examined by Dr. I. R. Pearson, who detected a mass in the left iliac fossa and instituted conservative treatment for probable pelvic abscess. He was examined by the writer about an hour later, when he seemed a good deal better, and no definite mass could be detected. Conservative treatment was therefore continued, but next day signs of intestinal obstruction developed and operation was decided upon. Under local anæsthesia, supplemented with thiopentone given intravenously and nitrous oxide and oxygen inhalation, the abdomen was opened by a left paramedian incision; what appeared to be a completely gangrenous Meckel's diverticulum was revealed, with gross œdema of the related bowel, causing intestinal obstruction. Three inches of gut including the gangrenous mass were excised, and bowel continuity was restored by side-to-side anastomosis. The abdomen was closed without drainage. Continuous gastric suction and intravenous drip saline therapy were instituted and continued for three days, by the end of which time borborygmi were heard and the patient had passed flatus spontaneously. Thereafter convalescence was uneventful, and he was discharged from hospital on the ninth post-operative day, apparently well.

The specimen was examined by Dr. M. P. K. Shoobridge at the Launceston General Hospital, who reported the presence of a diverticulum lined with normal mucous membrane, but terminating in a gangrenous mass. On examination of sections, the mass proved to be an extremely anaplastic carcinoma. Dr. Shoobridge was unable to state whence the tumour arose, but concluded: "A possible explanation (although rather a long shot) is that the tumour arose from ectopic tissue in the wall of a diverticulum—? Meckel's diverticulum." Shortly after the receipt of this report, and eight days after his discharge from hospital, the patient was readmitted with complete left hemiplegia, and was very ill for about ten days. He was examined by Dr. J. Grove, consultant physician, who diagnosed probable malignant embolism, and the patient was allowed home forty-six days after his readmission to hospital, with a hopeless prognosis. Three weeks later he died at home; but permission for post-mortem examination was refused, so the final diagnosis must remain in doubt. The original condition appears to have been acute intestinal obstruction secondary to acute gangrenous Meckel's diverticulitis, complicating anaplastic carcinoma which may have arisen primarily in the diverticulum.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Outlines of Industrial Medicine, Legislation, and Hygiene", by James Burnet, M.A., LL.B. (London), M.D., F.R.C.P.E.; Second Edition; 1953. Bristol: John Wright and Sons, Limited. 7½" × 5", pp. 122. Price: 10s. 6d.

Intended merely as an introduction to the study of the subject by students of industrial medicine and others.

"Furneaux's Human Physiology", by William A. M. Smart, M.B., B.S. (London), M.R.C.S. (England), L.R.C.P. (London); New Edition; 1953. London: Longmans, Green and Company. Melbourne: Longmans, Green and Company. Nurses Edition. 7½" × 5", pp. 426, with 165 illustrations. Price: 13s. 6d.

Intended for the use of nurses in training.

"Manual of Urology", by Alec W. Badenoch, M.A., M.D., Ch.M. (Aberd.), F.R.C.S.; 1953. London: William Heinemann (Medical Books), Limited. 10" × 7½", pp. 564, with 343 illustrations. Price: £5 5s.

"A practical manual", based on the author's own experience, which in turn is based on the traditions of Saint Peter's Hospital for Stone.

"Medicine in Oxford: A Historical Romance: The Fitzpatrick Lectures for 1952-53, Delivered before the Royal College of Physicians of London", by Maurice Davidson, M.A., D.M. (Oxon.); 1953. Oxford: Basil Blackwell. London: Macmillan and Company, Limited. 8½" × 6", pp. 78, with 13 illustrations. Price: 10s. 6d.

No additions have been made to the manuscript since the lectures were delivered.

"Sex in History", by G. Rattray Taylor; 1953. London: Thames and Hudson. 8½" × 6", pp. 344. Price: 21s.

The work is "more than just a history". . . . "above all, it seeks to show the remarkable continuity of the sex attitudes which form part of western culture: the proportions in which the elements are mixed vary widely, but the ingredients remain amazingly constant".

"Bodily Changes in Pain, Hunger, Fear and Rage: An Account of Recent Researches into the Function of Emotional Excitement", by Walter B. Cannon, M.D., S.D., LL.D.; Second Edition; 1953. Boston: Charles T. Branford Company. 8" × 5½", pp. 420, with 42 text figures. Price: $5.00.

Based on work carried out at the Harvard Physiological Laboratories.

"Australia in the War of 1939-1945: Series I, Army: Volume II." "Greece, Crete and Syria", by Gavin Long. Canberra: Australian War Memorial. Sydney: Angus and Robertson, Limited. Obtainable at all booksellers. 9½" × 6½", pp. 592, with 72 illustrations, seven maps in colour and about 80 sketch maps. Price: 25s.

A review of Volume I of this series was published in the issue of August 15, 1953.

"Physiological Foundations of Neurology and Psychiatry", by Ernst Gellhorn, M.D., Ph.D.; 1953. Minneapolis: The University of Minnesota Press. Geoffrey Cumberlege: Oxford University Press. Melbourne: Oxford University Press. 9" × 6½", pp. 570, with 107 illustrations. Price: 89s. 3d.

The author discusses some of the physiological foundations of neuropsychiatry with which he is familiar through his own experimental work.

The Medical Journal of Australia

SATURDAY, APRIL 3, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE DEATH OF DR. NORMAN GERALD HORNER.

THE death of Dr. Norman Gerald Horner, for eighteen years its Editor, is announced in the *British Medical Journal* of March 13, 1954. Horner was a gifted journalist and his editorship covered the difficult years 1928 to 1946. By his devoted service to the British Medical Association he brought his talents to the advancement of the medical sciences, and placed British medicine in his debt. He succeeded Sir Dawson Williams, who had been Editor for thirty years and who had occupied other positions on the staff for seventeen years before he became Editor. Horner's task on his assumption of office was not less difficult on this account. He had, however, acted as assistant to Dawson Williams from 1917 when he returned to England from military service in France. He had also, prior to his joining the Royal Army Medical Corps, served on the editorial staff of *The Lancet* with the late Sir Squire Sprigge. His training had fitted him for his responsible position, and the obituary notice in the *British Medical Journal* shows that he had much to contend with at first in the matter of staff shortage and illness. We read that he thought that the Association "did not fully appreciate the difficulties under which he was then working". He was probably right, because few members of the medical profession have any idea of what is involved in the running of a journal even in normal times, and fewer still try to learn anything about it. Horner was a good friend to THE MEDICAL JOURNAL OF AUSTRALIA—he gave useful advice when he was asked for it, and on the occasion of the Silver Jubilee of the Journal in 1939 he sent a generous message of appreciation for publication in the Jubilee Number. After his retirement from the editorship of the *British Medical Journal* in 1946 Horner joined those who collaborated with Sir Arthur S. MacNalty in the preparation of Britain's "Official History of the War of 1939-1945". He was a reserved and apparently shy man; he was gentle and courteous, but one felt that beneath the calm surface lay a fund of energy and tenacity of purpose. THE MEDICAL JOURNAL OF AUSTRALIA joins with others in the Old Country in paying a tribute to his memory—to his personal worth and the value of his work.

SOME DIFFICULTIES OF MEDICAL PRACTICE IN TASMANIA.

THERE are some differences of medical practice in all the Australian States, but medical practice in Tasmania is unlike that of any of the other States. This means that the British Medical Association in Tasmania, and in particular the Council of the Tasmanian Branch, are often faced with problems of peculiar difficulty. It will not help our present situation to review the history of Tasmanian medical practice as it existed twenty-five or thirty years ago. If we did so, it might do two things. First of all, it might show the members of the Tasmanian Branch that they have to hasten slowly in coming to important decisions, and it would probably also impress on them, as indeed it should on all inhabitants of the island, that the Tasmanian Government can and does do things which, in the long run, are not for the good of the health and well-being of the Tasmanian people. Our present object is to bring to the notice of members of the profession the peculiar position in which members of the profession in Tasmania are placed in regard to hospital practice.

Some few weeks ago, an advertisement arrived from the Director-General of Medical Services of Tasmania in which applications were invited for registered medical practitioners for part-time appointments. Four officers were needed—a physician, an obstetrician and gynæcologist, an oto-rhino-laryngologist, and an oculist. It was stated that applicants would be required to possess a recognized higher qualification in their particular specialty. The appointments required successful applicants to devote five sessions per week (each session to be of four hours) to work in public hospitals on the north-west coast of Tasmania controlled by the Department of Public Health. It was stated that private practice was allowed and that remuneration was to be at the rate of £1500 per annum. On the face of it, this advertisement appeared to be satisfactory because the sessional payments were adequate and private practice was allowed. The Tasmanian Branch Council, however, pointed out that, in its opinion, the advertisement was misleading. In Tasmania, the State Government controls the public hospitals; there is a large general hospital in Hobart and one in Launceston, and smaller hospitals are found at the main country centres. The only private or intermediate beds available in any hospitals are in Hobart and Launceston. No private or intermediate beds are provided in any public hospital. All government hospitals are open to anyone in the community regardless of their income—there is no application of the means test. The Tasmanian Branch Council took the view (and there is no doubt that its view was correct) that no specialist of the type required in the advertisement could be expected to carry on a proper private practice

unless he had private and intermediate beds to which he could admit his patients. The advertisement was misleading in that it really offered something with one hand and took that something away with the other. Any request that the Tasmanian Government should establish private or intermediate beds, whether that request comes from the Tasmanian Branch of the British Medical Association or from medical practitioners in the State or any other source, is met with a curt refusal because any compliance with such a request would be contrary to Government policy. This matter has been given a great deal of publicity in Tasmania and this journal has been censured for its refusal to accept the Tasmanian Government's advertisement. In fact, most of the statements made here have been set out in the Press.

Readers of this journal will probably recall that it is part of the Federal Council's policy that members of the Association should not act as honorary officers to hospitals which do not apply the means test. The Tasmanian Branch has not been able to fall into line with this policy because the members of the visiting specialist staff in Hobart and Launceston find themselves in a peculiar position. For some years they were paid for attendance on a sessional basis; but about two years ago this payment was stopped, probably as an economy measure on the part of the Tasmanian Government. It should be pointed out that there are several part-time paid specialists in Hobart and Launceston. These officers can, of course, conduct private practices because there still remain some private hospitals in both these centres. The Government, we understand, agrees in principle that sessional payments should be made to visiting staffs in Hobart and Launceston, but they do nothing about adhering to that principle. At the same time, although it has no money to pay for sessional service in Hobart and Launceston, it has enough money to try to induce part-time specialists to settle in particular areas of the island. The position in Hobart and Launceston is absurd, because by giving honorary service to all people in public hospitals, regardless of their incomes, the specialists are really competing against themselves in private practice.

This whole matter is, of course, bound up with the freedom of the individual. Everyone will agree that the people in north-western Tasmania are entitled to, and should have, specialist medical service available to them. At the same time, they should be able to have this service in the way in which they would prefer to have it, and they should be allowed the opportunity of private and intermediate hospital service if they desire it. The matter of "policy" can be overdone, and we have no doubt whatever that the Tasmanian Government is overdoing it.

Current Comment.

CONTACT DERMATITIS AND PETROLEUM PRODUCTS.

Petroleum products are used widely and in great variety in modern industry, but they bring their own problems, notably in the form of dermatitis. According to N. P. Anderson,[1] the cutaneous reactions occurring as a result of exposure to petroleum products may be considered in four or five main groups. Of these, primary irritation is probably the most common. It is not a form of contact (allergic) dermatitis from a sensitizer, but rather dermatitis due to repeated and prolonged contact with degreasing or defatting agents, including the so-called solvents. These include kerosene, petroleum ether, gasoline, naphtha, crude benzine, tetrachlorethylene and similar products. The skin condition comes on gradually and may be complicated by infection with pyogenic organisms, leading to boils, carbuncles, impetigo, echthyma, cellulitis, lymphangitis and infectious eczematoid dermatitis. Anderson points out that some solvents are frequently used by workers for the cleansing of the skin from oils, greases and dirt. The effect on the skin of such solvents as kerosene, benzene and the rest is one of defatting. The exposed skin gradually becomes excessively dry, wrinkled and somewhat scaly, and finally red, roughened and chapped. The second condition seen as a result of contact with petroleum products is that of sensitization or allergic dermatitis, which produces an eczematous reaction. More than one exposure is necessary, and there is a period of incubation, which may be as short as ten to fourteen days. However, it may not occur until the worker has been exposed for months or even years. The development, in many cases, is facilitated by the prior accidental occurrence of a scratch, cut or other break in skin continuity. The areas most frequently affected are the hands and forearms, followed by the face, eyelids and neck. The condition begins with redness, superficial papules and vesicles, and the process tends to be diffuse. Interdigital maceration with small vesicles on the sides of the fingers and the ulnar border of the hands may be present. The bends of the elbows are often affected. So-called soluble cutting oils are commonly responsible. A third great problem is that of oil acne and folliculitis. This condition occurs not only in oil and refinery workers, but also in machinists who are exposed to heavy oils and to chlorinated hydrocarbons. It is thought to be due to primary blocking of the pilo-sebaceous openings or to chemical irritation of the follicles. The chemical picture may vary from the presence of multiple blackheads to the development of follicular papules and papulo-pustules, and tends to involve the more hairy regions. The presence of metal splinters and dirt, the use of emulsions of mineral oils and alkalis, and uncleanliness of workers are all contributory factors to the occurrence of oil acne and folliculitis. Prophylactic treatment consists primarily of protective measures involving both worker and machine. Anderson states that wax boils are probably due to a combination of mechanical blocking of the pilo-sebaceous follicles and specific irritation caused by unrefined wax. He goes on to refer to certain petroleum products which may possess photosensitizing properties and states that photosensitization is most apparent with the cruder materials. While sensitization to gasoline occurs, it must be noted that it is a primary skin irritant. In some cases it is not the gasoline which is the cause of the cutaneous condition, but extraneous chemicals added, such as tetraethyl lead and various oil-soluble dyes used in colouring gasoline. "Diesel dermatitis" is due to chromates which have been added to diesel oil, usually in the form of sodium chromate. Anderson quotes the particular instance of workers who had become sensitized to chromate, which was added to prevent corrosion. They suffered from repeated attacks of eczematous dermatitis involving the hands and forearms, and were found to react positively to patch tests with 0·5% sodium chromate solution.

[1] *Indust. Med.*, June, 1953.

POLIOMYELITIS IN TEXAS.

An epidemic of poliomyelitis in Hidalgo County, Texas, was carefully studied in 1948, and, although five years elapsed before the findings were published, the interest remains, especially as regards the mechanism of spread. Two years previously, investigations were carried out on the control of diarrhœal disease by insecticides in nine towns in Hidalgo County, and as the fly population had

access to privies, a fall in the incidence of dysentery was to be expected after treatment of this source of infection. This hope was realized, as infections by the Shigella group were reduced in number, and the fatal cases also. Early in 1948 poliomyelitis became prevalent for the first time in the county, and in view of the known excretion of the virus in the fæces it seemed worth while to try to reduce the density of infection of poliomyelitis by similar means. The presence of two ethnic groups in the county, Latin-American and Anglo-American, added to the interest of the epidemiological studies. The patients were examined in a hospital ward specially established for the study of this disease, and the work was carried out by Ralph S. Paffenbarger under the ægis of the United States Department of Health, and James Watt, from the Poliomyelitis Laboratory, Baltimore.[1] Population figures of the nine largest towns were checked, and rural figures estimated. Detailed data were collected by special workers so as to gain information about homes as possible source of infection. Careful definitions were drawn up to establish criteria of infection and of "probable" infections, and adequate descriptions of "contact", both direct and indirect, were accepted. These criteria were rigid, and in the cause of accuracy other possible but unproven avenues of contact were disregarded. One point of interest was noticed. During the last forty years some changes have occurred in the age-specific attack rates in the United States, the previously held figure of under five years of age, from 9% to 12% of total population, no longer furnishing 50% to 90% of the cases with the same consistency. This reckoning, however, still held good for Hidalgo county, no doubt owing to the relative immunity of this area during previous years. One question which the workers wished to solve was the relative importance of human beings and flies as vectors of the disease. Fly counts were carried out and the figures were compared between the "treated" areas and others. Relations of the fly populations were also studied in different areas. With regard to the possible human vectors contact within households and contact in the general population were compared as far as the information was obtainable. When the evidence was examined, little was in it to suggest that flies played any significant role in spreading the epidemic. The authors admit that even in "treated" areas only a certain proportion of the flies were killed, but the estimation of the importance of this consideration is like the reliability of a history of human contact, very difficult to establish. An effort was also made to determine the significance of non-paralytic infections; the figures available show that the rate per 1000 of Anglo-Americans was 197 and that of the Latin-Americans 34. The authors sum up their findings by stating that fly control instituted before and continued during an outbreak failed to reduce numbers of cases or "to affect the time-course of the epidemic in cities with community-wide fly control". The number of histories obtained in which contact was established with a preceding paralytic case is suggestive of this being the mechanism of transfer. Finally they think that personal contact, directly or indirectly, played a "definitive role" in the spread of the epidemic. In this last study the presence of the two ethnic groups with different socio-economic conditions was helpful in the analysis.

As a pendant to this inquiry Joseph L. Melnick and Richard P. Dow[2] studied the occurrence of poliomyelitis and Coxsackie viruses in flies in the areas under surveillance. They found a correlation between the finding of poliomyelitis in flies and privy specimens collected in the same area, and found that this virus was transmissible to monkeys. This virus was more frequently found at the peak of the epidemic. Coxsackie virus was also found in mixed antigenic types. Thomas Francis, junior, Gordon C. Brown and John D. Ainslie demonstrated both Coxsackie and poliomyelitis viruses in privy specimens.[3] Since virus is found in the fæces of persons with either clinical or subclinical forms of poliomyelitis, such studies could be taken as a measure of the spread of an epidemic. The details of this research are not simply summarized, but it may be mentioned that subclinical infections were a feature of one area where virus was found, though no overt case was seen. There are still obscure problems concerning the virus, its habits and activities both within the human body and outside it.

[1] *Am. J. Hyg.*, November, 1953.
[2] *Ibidem.*
[3] *Ibidem.*

STINGRAY INJURIES.

IT appears fortunate that the stingray is a somewhat indolent creature and that its venomous propensities seldom constitute a real danger to man. Injuries by stingrays may be caused by incautious handling by a net fisherman or by treading on one of these fish lying partly buried in mud or awash on sand. The Australian literature contains several accounts of the venomous equipment of the stingray and its general habits and mode of attack. The contributions of G. P. Whitley should be mentioned, and clinical accounts of J. B. Cleland and W. E. Paradice, these last two being published in this journal in 1942 and 1944 respectively. Cleland shows how severe and deep the wound may be when inflicted by a stingray ten feet in diameter, though it would seem more likely that the unwary wader should tread on a small than a large specimen. Findlay E. Russell, writing from Pasadena, California, quotes these Australian authors among his 47 references, and collects in an article the known information about the stingray and gives case notes of four patients who were injured in an attack.[1] The maximum weight is said to be in excess of 700 pounds, but off the Californian coast the commonest type is about 50 centimetres in length, with a sting of six centimetres. Writers on the subject go back to Aristotle as an authority, but sure knowledge concerning the nature of the venom gland and the mechanism of the attack has come only in recent years. The author quotes the work of H. M. Evans, who in 1916 described specialized secretory cells lying along the lateral grooves on the ventral surface of the sting. When an intruder disturbs the stingray, as, for example, by treading on its back, it immediately reacts by thrusting the tail and sting forward and upward into the lower extremity of the victim. The stingray does not appear to be aggressive except in response to disturbance. However, injury may occur in two ways, by the forceful thrust of the caudal organ, and by the introduction of venom from the poison gland. The sting itself is calcified, with serrations along each side, with teeth (using the word in a general sense) so curved that the withdrawal of the sting will cause lacerations. This may cause a severe and painful wound; in the case quoted in Cleland's account the wound penetrated the space between the tibia and the fibula, causing a wound seven inches long on the lateral aspect. It would be expected that such a wound would be jagged and need surgical attention. Pain is naturally a feature of all severe wounds, but it is also due to the poison, whose secretion by the epithelial organ has been proved, not only by clinical observation but also by experimental work. The author has never seen paralysis of muscles, but this has been described by other observers. Pain may be slight or severe; cramps may occur and acute abdominal pain with diarrhœa. Russell by personal inquiry from 29 patients investigated has found that the majority complained that the pain was one of the most severe they had ever experienced. Shock and sweating were also observed. It would seem that there is a general resemblance between all the types of symptoms occurring after wounding inflicted by fish of different kinds possessing poisonous spines. One other sign may be mentioned—local œdema, which may persist for variable periods. When the swelling begins immediately it would seem reasonable to attribute it to the local effect of the venom, but when it lasts for some time and is accompanied by other signs suggesting inflammation it would surely be due to infection. The muddy slime contaminating the sting might easily carry in bacteria. This feature of these injuries has been noted by a number of writers and calls

[1] *Am. J. M. Sc.*, December, 1953.

for careful surgical cleansing. *Débridement* has been recommended, but it would seem wise not to draw adapting sutures tight if these are deemed appropriate for use. The most important items of treatment are the alleviation of pain, for which morphine or substitute will probably be necessary, and measures directed towards prevention of secondary infection. Whether anything can be done to neutralize the effect of the venom or not is doubtful. The literature contains a number of remedies, which, as might be expected, range from suggestions with very little scientific basis to frank witch-doctoring. The sea contains a number of enemies to man, and the stingray is poisonous enough to warrant careful handling of its wounds on sound medical and surgical lines.

HOW CONSTANT IS THE I.Q.?

The intelligence quotient (I.Q.) is used extensively in various fields to provide an objective assessment of an individual's intelligence. It is accepted in medical, educational, social and commercial fields, and sometimes a good deal is allowed to depend on it. Just the same, it is never wise to depend too much on the result of a single test, as is, or should be, well recognized in relation to such things as clinical pathological examinations, and it is well to realize that assessment by means of the intelligence quotient has distinct limitations, particularly when it is used for predictive purposes. This has been made clear by two psychologists, A. D. B. Clarke and A. M. Clarke.[1] There is no doubt, they point out, that intelligence tests provide a method more valid than subjective or clinical judgement in assessing a person's intellectual level. However, the additional assumption has developed that this level remains sufficiently constant during the life of the individual for fairly accurate predictions to be made about his future ability. In other words, a person's intelligence at, for example, the age of five years would be the same relative to others of his age group at later ages; thus the young genius would always be a genius, the average child would always be average, and the defective would remain defective. Such, these two psychologists state, are the implications of a belief in a constant I.Q., a belief which, within broad limits, seems to be in accord with common observation. Unfortunately, the belief appears to be unsoundly based. Many references are given in this paper to studies of I.Q. constancy in the United States, in which the concept of a rigidly constant I.Q. is contradicted by the facts. Indeed, I.Q. constancy over long periods of time during the years of mental growth appears to be the exception rather than the rule. These studies relate to mental growth in normal people in the general population. From this point Clarke and Clarke have gone on to ask the questions: "How permanent is intellectual subnormality? Is 'once a defective always a defective' true?" From carefully controlled tests of over 100 adolescent and adult certified defectives, they were able to find considerable and significant variations in the I.Q. level over a period of time, the changes being largely in the upward direction. They examined a number of hypotheses advanced to account for the increase in I.Q., but rejected all except one—namely, that there was a strong relationship between very adverse early home conditions and subsequent I.Q. increases. They find it consistent with their findings to suggest that the environment which is really antagonistic towards the child retards mental development for many years. Later, however, after removal of the child from such conditions, this retardation begins to fade and I.Q. increments occur, often at ages when mental growth is commonly assumed to have ceased.

These findings have important implications. It is obviously fallacious and unfair to a child to make a permanent plan for a child's future or allocation to a particular type of activity or training on the basis of a single mental test. Certainly, the subsequent assessment of a child's progress and attitude towards his work should be made in the light of his currently determined intelligence quotient, and not on that originally recorded. If this is not done, the initially brilliant child can be readily thought to have failed to make the most of his opportunities; and on the other hand, the apparently mediocre child may be deprived of subsequent opportunities which he is well qualified to use. That is the position for the children of normal mental outlook. So far as those of an apparently defective mental outlook are concerned, Clarke and Clarke mention one important point as an example. On the basis of the finding that a group of socially deprived mentally subnormal people tend, when removed from adverse conditions, to advance towards intellectual normality, they ask whether such relatively spontaneous changes can be accelerated and improved. The question is important, and warrants a thorough investigation.

[1] *Lancet*, October 24, 1953.

MONEY: A REHABILITATION INCENTIVE FOR MENTAL PATIENTS.

Psychiatrists in recent years have become increasingly concerned with the rehabilitation of mental patients. Peter A. Peffer[1] suggests that two factors contribute to this attitude: first, the goals of psychiatric treatment exceed the horizons of the private consulting room or the mental hospital; second, there are rarely enough hospital beds for the patients who need them. Peffer states that to meet the ethical, philosophical and practical demands of psychiatry it is essential that effective rehabilitation techniques be developed that will enable mental patients to return to society as satisfactorily functioning individuals. He quotes Webster's definition of rehabilitation as a process "to restore a person, as a disabled soldier, to a station of independent earning power", and points out that in this definition we find no compromise, no reduction of goals to ineffective levels. The optimum of rehabilitation is not the ability to make an occasional basket, the copying of magazine pictures, the creation of a leather purse or the carving of book ends. These have their place in the rehabilitation process, but they are only means to an end. Few people could maintain "a station of independent earning power" by such activities. Rehabilitation must mean a great deal more. Patients can master almost every skill provided in occupational therapy and manual art shops of mental hospitals and still remain permanent residents of the chronic wards. Peffer suggests that much of the difficulty involved is the result of failure to think of the mental hospital as part of society, and so to develop the means of transition from one to the other. He considers that any technique intended to help bridge the gap must take into account the need for an incentive reward system with factors common to the hospital and society. Recent work has indicated that the fundamental goals and incentives of a culture pattern have a motivating power equal to innate drives, whereas there is little incentive value in goals not consistent with the individual's cultural pattern. Research on values and incentives in the American culture has revealed one major incentive that forms a fundamental foundation of the American way of life; that incentive is money. There is some experimental evidence that the monetary incentive incites more physiological, social and personal motives in Americans than any other incentive in their culture. Whether one regards that as a good thing or a bad thing, it is apparently the fact and has to be acknowledged when a rehabilitation incentive is being sought. Moreover, as Peffer points out, it is an incentive that is as effective within a hospital as it is outside. It is not suitable for all mental hospital patients, but it has a wide application and provides a bridge over the gap. The point is that even within a mental hospital no administration will provide a money reward system for nothing. Even for a psychotic patient to receive payment, he must produce something that is valuable to society. Money reward can be obtained only by a work pattern that is normal. Thus,

[1] *Am. J. Psychiat.*, August, 1953.

the patient in the mental hospital comes to do, at his own level, what people do outside and receives the same kind or class of reward as the people outside receive. As an experiment, Peffer managed to have extended to mental hospital patients a system, long established in domiciliary centres of the Veterans Administration, in which those undergoing rehabilitation are described as "member employees". Those elected as member employees are first discharged from hospital as patients and then housed in the staff quarters. They receive board, but have to provide clothing from their own earnings. The rate of payment depends on the job classification and the hours worked. With a successful work record, it is much easier for the person concerned to obtain a position outside the hospital. Peffer states that to date 20 mental hospital patients have been fitted into this scheme, and only three have been readmitted to the hospital. Remarkable personal adjustments have resulted, with improvements in appearance, behaviour and self-esteem. It has also been a considerable financial saving to the Government. The system has given current occupational therapy and manual arts procedures a higher goal and purpose. It provides for the placement officer a recent work history to facilitate his task with industrialists in placing the former patients in outside industry, and it permits former mental patients to be hired on the same basis as normal persons. The system has difficulties and limitations which Peffer discusses, and its full application is still being worked out, but so far, apparently, it has proved to be a most effective intermediate extramural rehabilitation process.

THE FIRST APPENDICECTOMY.

VARIOUS VIEWS are current about when the first appendicectomy was performed, but it appears that the credit must go to Claudius Amyand, principal surgeon to Saint George's Hospital, London, for removing the appendix of a boy during the cure of a scrotal hernia on December 6, 1735. Philip G. Creese[1] quotes Amyand's full account of his operation published in the *Philosophical Transactions of the Royal Society* in October, 1736. Apparently, Amyand found the operation the most complicated and perplexing he had ever met with, "many unsuspected Oddities and Events concurring to make it as intricate as it proved laborious and difficult". The appendix was in the hernial sac and was perforated by a pin, with a resultant fistula and intermittent discharge of fæces. The appendix was amputated and an attempt made at repair of the hernia. Amyand comments that he took a long time over the operation, almost half an hour, although, as Creese points out, the speed of the operation seems remarkable to us. It must certainly have seemed exceedingly slow to the patient in the absence of anæsthesia; and from Amyand's description of what went on, it is apparent that, as he remarks, "the Patient bore it with great Courage". The hernia recurred, which is not remarkable, but the recovery of the patient from his operative ordeal was certainly a notable achievement. Apparently Amyand sent the surgical specimen to the repository at the Royal Society, but it is no longer in existence. Creese discusses at some length the history of appendicectomy and the various claims made about those alleged to have performed it first, but Amyand's claims to priority are probably valid. To complete the picture, Creese has managed to get together a remarkable amount of biographical detail about Amyand, although, as he says, it is a collection of bits and pieces; unfortunately there is no complete extant record of Amyand's career, nor is there any picture of him known to be in existence. Amyand had, in fact, a number of claims to fame. He was Sergeant-Surgeon to King George II, a pioneer in smallpox inoculation, a Fellow of the Royal Society, the first principal surgeon to the Westminster Hospital, a founder and first principal surgeon to Saint George's Hospital, Master of the Barber Surgeons, which made him one of the leading London surgeons of his day, and the first surgeon to perform an appendicectomy. For all that, he has remained in obscurity. This is a pity, for, as Creese remarks, Claudius Amyand was not a man of genius, but one of solid worth who merits a nod of recognition from medical history too long denied him.

[1] *Surg., Gynec. & Obst.*, November, 1953.

SERUM POLYSACCHARIDES IN ARTHRITIS.

MANY of the proteins in blood serum are combined with polysaccharides of varying degrees of complexity. Variations in the amount of polysaccharide combined with a particular protein occur, but little if anything is known of the reason for this. The increase in serum polysaccharide content in certain types of arthritis has been considered to be related to increases in tissue polysaccharide content in these diseases.

A more complete analysis of the characteristics of these serum components in rheumatoid arthritis, certain other mesenchymal diseases and degenerative arthritis has been made by M. R. Sheflar, R. W. Payne, J. A. Bullock, D. R. Patrick, A. A. Hellbaum and W. K. Ishmael.[1]

They used the method of Milne with sodium sulphate to separate albumin, euglobulin and pseudoglobulin from the serum. Mucoprotein was also separated. Polysaccharide content was determined in the total protein and each of the fractions. Elevations of the polysaccharide content of total protein, albumin, pseudoglobulin and mucoprotein were found consistently in the sera of patients with rheumatoid arthritis, but not in those with degenerative joint disease. The polysaccharide content of the pseudoglobulin fraction was found to be elevated in inactive rheumatoid arthritis, while other components were unchanged. No significant correlation between erythrocyte sedimentation rate and the polysaccharide percentage of serum protein was found in patients with rheumatoid arthritis. Sedimentation rates were generally elevated in the case of patients with rheumatoid arthritis, but many of the patients with degenerative joint disease also had elevated sedimentation rates. The authors suggest that the determination of serum polysaccharide content might be of value as a diagnostic aid to distinguish degenerative joint disease from rheumatoid arthritis and also as an objective test for evaluating the degree of activity in patients with rheumatoid arthritis.

[1] *J. Clin. Investigation*, December, 1953.

A PLAQUE TO THE MEMORY OF THE LATE WILLIAM JOHN HANCOCK.

A PLAQUE to the memory of the late Dr. William John Hancock has been erected at the Australian Institute of Anatomy, Canberra, A.C.T. The inscription is as follows.

HANCOCK, WILLIAM JOHN D.Sc., M.I.C.E., M.I.E.E.,
1863-1931 Hon. M.I.E.E.
(PERTH)

A Pioneer in Radiology who died as a result of the injuries sustained in his researches.

He was Hon. Radiologist to Perth Hospital for 22 years and Hon. Consulting Radiologist until his death.

He was a foundation member of the Röntgen Society London and an Hon. Member of the British Medical Association (W.A. Branch).

He was elected a member of the Senate of the University of West Australia at its inception and re-elected until his retirement. He was awarded the "Kelvin" Gold Medal for 1926 by the Royal Society of West Australia.

INDEX TO "THE MEDICAL JOURNAL OF AUSTRALIA".

THE index to THE MEDICAL JOURNAL OF AUSTRALIA for the half-year ended December 31, 1953, has been published. Readers who are on the index mailing list will receive their copies as usual. Others who desire to receive a copy should apply to the Manager at The Printing House, Seamer Street, Glebe, New South Wales.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

An Enzyme in Inhibition of Growth of Tubercle Bacilli by Spermine.

JAMES G. HIRSCH (*J. Exper. Med.*, March, 1953) investigated the essential participation of an enzyme in the inhibition of growth of tubercle bacilli by spermine. If the bovine albumin in the medium to show the action of spermine was replaced by crystalline albumin, the activity disappeared. However, beef and sheep serum could replace the bovine albumin. An attempt was then made to discover what portion of these substances acted as an activator to spermine against tubercle bacilli. It appeared that the substance had the property of an enzyme which acts on spermine and whose product exerts a toxic effect on mammalian tubercle bacilli. The substance appeared to be part of the α globulin fraction.

Induced Malignancy in Cells.

HARRY GOLDBLATT AND GLADYS CAMERON (*J. Exper. Med.*, April, 1953) observed induced malignancy in cells from the rat myocardium subjected to intermittent anaerobiosis during long propagation *in vitro*. The rat was one of a colony which had been tumour-free during a considerable period of study, and the tissue culture had been carried through a number of transplants in an atmosphere of nitrogen. The change of the cells from the normal spindle shape and the occurrence of abnormal mitosis indicated the morphological change, and on implantation of fragments of the culture into normal rats, tumours developed, in situations such as in the subcutaneous tissues of the flank and in the eye. Microscopic examination of the tissues confirmed their malignant character.

Egestion of Phagocytized Particles by Leucocytes.

ARMINE T. WILSON (*J. Exper. Med.*, October, 1953) employed phase-contrast microscopy to study the process of phagocytosis and noted egestion of phagocytized particles by leucocytes, a phenomenon which appears not to have been commented on previously. Cultures of hæmolytic streptococci were grown for two or three hours in Todd Hewitt broth with 10% normal rabbit serum added; they were then centrifuged, and the deposit was taken up in fresh broth. Fresh human blood was treated with treparin solution 1:10,000, and a loopful of blood and a loopful of organisms were mixed on a coverslip; a hanging drop was then made and placed in a warm chamber attached to a phase-contrast microscope for study. Photographs were taken at intervals. Successful preparations can be observed for several hours. It was noted that when a coccus was ingested, it first appeared in a vacuole, which might persist until the cell died. However, in some instances the cocci were immediately ejected; they trailed beside the leucocyte and were later ingested again. Some cocci were seen to divide after having been inside a leucocyte. The ability to divide seemed to be lost if the coccus had been within the leucocytic vacuole for a long period of time. The author discusses the significance of this phenomenon and whether it is of importance *in vivo*.

Interaction of Antigens and Antibodies.

J. R. MARRACK AND R. A. GRANT (*Brit. J. Exper. Path.*, June, 1953) made quantitative studies on the interaction of antigens and antibodies in the presence of low concentrations of salt. Their technique was developed on the observation that in solutions of protein that have the same concentration by weight, a beam of monochromatic light will scatter at an angle of 90° to the incident beam an amount of light that is approximately proportional to the size of the molecules. The scatter of solutions of globulin prepared from various types of albumin, α globulin and pepsin was established; then varying amounts of salt were added to the solutions, with the corresponding antigens, and the amounts of precipitate and the reduced density of the solutions were measured. It was found that the pH of the mixture had a considerable influence on the amount of sodium needed to cause flocculation; in solutions with low sodium concentrations, although the combination of antigen with antibody took place, the solutions remained clear—that is to say, in low sodium concentrations the visual effect of the antigen-antibody combination is lost.

Vaccination Against Influenza B with Monovalent Vaccine.

A. V. HENNESSY, ELVA MINUSE, F. M. DAVENPORT AND T. FRANCIS, JUNIOR (*Am. J. Hyg.*, September, 1953), describe an experience with vaccination against influenza B infection in 1952 by use of monovalent vaccine. The subjects were children in a training school for high-grade mental defectives, and they were divided into three groups. One group was given polyvalent influenza virus vaccine type A, another influenza virus vaccine type B, and the third a saline control vaccine. At the time an epidemic appeared the groups were numerically nearly equal—219, 207 and 212 respectively. Significant protection against type B infection was clearly demonstrated in the vaccinated group, and cases occurred chiefly in the low antibody level subjects in both control and type B vaccinated groups. The incidence of type B influenza was more frequent in younger children than in older ones. Antibody levels were estimated six months after vaccination, and were found to have fallen considerably. The authors concluded that lack of experience with the antigens of type B virus was responsible for the low level of antibody in the younger children and that they were selectively attacked by the disease, thus demonstrating that susceptibility is a function of age.

Serum Antibodies in Mumps.

B. GHOSH RAY AND R. H. A. SWAIN (*Brit. J. Exper. Path.*, October, 1953) state that recognition of the fact that meningo-encephalitis due to mumps virus may occur without enlargement of the salivary glands, and during a time when poliomyelitis is also prevalent, makes it necessary to distinguish this from other types of meningo-encephalitis. They examined patients with typical parotitis during an epidemic, in order to compare the results of complement-fixation tests and hæmagglutination-inhibition tests with virus-neutralization tests on the patient's serum. It was found that the antihæmagglutinating antibody did not rise to a maximum for twelve days from the onset, but could be demonstrated for many months with little loss, and the neutralizing antibody behaved similarly. The complement-fixation reaction, on the other hand, appeared as early as the second day in relation to both soluble and infective components, but this reaction disappeared relatively rapidly. The latter test then is of value for early diagnosis in atypical instances of the disease. Rise in antibody titre in three patients with meningo-encephalitis but no clinical signs of parotitis gave good evidence that the meningeal infection was due to the mumps virus.

Pathogenesis of Fever.

I. L. BENNETT AND P. R. BEESON (*J. Exper. Med.*, November, 1953) began a study of the pathogenesis of fever by testing the effect of injection of extracts and suspensions of uninfected rabbit tissues upon the body temperature of normal rabbits. The techniques used were designed to eliminate known sources of pyrogenic substances; glassware was carefully sterilized, biological materials were tested for pyrogenicity, and cultures were made in thioglycollate broth in order to control contamination. Extracts and suspensions from a wide range of rabbit tissues were prepared and, in the case of extracts treated with penicillin and streptomycin, filtered and stored at 4° C; while suspensions were ground in saline with antibiotics, shaken and centrifuged at 2000 revolutions per minute, filtered and stored. Intravenous inoculations into normal rabbits were followed by the recording of temperatures at short intervals for three or four hours. Two extracts of bone marrow produced rises of 0·9° and 1·5° C., but these were the only effects shown from this series of experiments. Another series of experiments was designed to produce infarction of such organs as kidney, spleen and portion of lung. Histological examination of the material later showed areas of hæmorrhagic necrosis, but in no case had fever been demonstrated. A third group of animals was tested for fever in response to the induction of the Schwartzman's reaction with intracutaneous injection of bacterial polysaccharide prepared from *Serratia marcescens*, followed by excision of the reacting skin and preparation of an extract; this extract was then injected into a normal rabbit by the intravenous route, and the temperature recorded. The extracts in the great majority of instances produced prompt rises in temperature in the test animals. The next series of experiments were made with extracts from polymorphonuclear leucocytes and from peritoneal exudate fluid, and here prompt rises in temperature were obtained, and the effect was abolished if the extracts had been heated to 90° C. for thirty minutes. Characterization of fever-producing substances from polymorphonuclear leucocytes and from the fluid of sterile exudates was then studied in further detail. First, extracts were made of leucocytes obtained from whole blood and tested, and again a heat-labile fever-producing substance was found.

Analyses were made to demonstrate the composition of the active substance; the protein and polysaccharides in fluid sufficient to evoke the febrile response were of the order of 0·76 microgramme of protein and 0·054 microgramme of polysaccharide, and enzymic digestion with trypsin or retonuclear did not remove the fever-producing property. Some of the known pyrogens, such as Menkin's pyrexin and bacterial pyrogens, were compared, and it was found that the tolerance developed to bacterial pyrogens did not alter the response to the extract of leucocytes. The use of amidopyrine suppressed the response to leucocytic extracts, but did not influence the response to peritoneal extracts. This led the authors to the conclusion that the leucocyte is not the only source of the factor.

HYGIENE.

Venereal Diseases: Present and Future.

N. J. FIUMARA (*Am. J. Pub. Health*, November, 1953) surveys the public health problem of the control of venereal diseases in the United States. He states that a great reduction in the incidence of syphilis and gonorrhœa has occurred as the result of efficient control programmes and treatment with penicillin, which quickly reduces the infectious period. The speed and ease of cure mean that reinfections are common. The author considers that attempts to get all the patient's contacts at the time of treatment should be made, and health departments should offer the services of trained interviewers to private practitioners for this purpose. Outpatient centres of general hospitals should be utilized for the care of the medically indigent. Here the contact-interviewers and investigators will have the opportunity to interview their patients. These clinics can also serve as consultation and teaching centres, as well as centres for applied research, particularly in diagnostic and treatment fields. The problem of sexual promiscuity has as yet to be solved. There need to be organized and co-ordinated efforts on the part of the home, church and community to train and fashion the character of people and particularly youth. If control efforts are relaxed and the present level of sexual promiscuity persists, one can safely predict epidemics of these diseases in the future.

An Outbreak of Infectious Hepatitis.

A. M. LILIENFIELD, I. D. J. BROSS AND P. E. SARTWELL (*Am. J. Pub. Health*, September, 1953) made an epidemiological study of an outbreak of infectious hepatitis in a housing project in Baltimore during 1951. Each house was connected to the city's water supply and sewerage system. A house-to-house survey of 90% of the 888 resident families was made. Over a one-year period the attack rate was 2·9%. Those in the age group five to fourteen years experienced the highest attack rate, and females had a somewhat higher rate than males. Comparison of age-adjusted frequencies showed no important differences relating to school attended, to income status of family or to duration of residence in the city, or according to whether or not parenteral inoculations had been given. Individuals who had recently moved into the project had a lower attack rate than others. Secondary attack rates were computed, based upon all cases occurring during the two months after appearance of an initial case. The secondary attack rate among those who had not received γ globulin was 8·8%, while one case occurred among the 74 who had received it. The unprotected secondary attack rate was lower in most reported outbreaks, but these were in rural communities. This difference, which is consistent with British reports, suggests a higher endemic level of infection in urban communities, resulting in a smaller number of susceptibles available for infection upon the occurrence of a focal outbreak. Study of the spatial distribution of the disease showed that persons in households immediately adjacent to an infected household had an increased risk of attack over those at a greater distance. On the basis of certain assumptions regarding differences in the pattern of distribution of diseases transmitted by the fæcal-oral route, as compared with the airborne route, it is considered that the present data are more consistent with the fæcal-oral route than the airborne route as the means of transmission of the virus.

Field Studies on Manual Dishwashing.

M. A. SHIFFMAN (*Am. J. Pub. Health*, December, 1953) reports the results of a field survey of the relative effectiveness of two and three compartment sinks used in the manual washing of dishes in public eating and drinking places. He states that the American Public Health Service recommends that where chlorine sterilization is used, a three-compartment sink should be provided. The dishes are washed in the first, rinsed in clean water in the second and immersed in chlorine solution in the third. This procedure is supposed to prevent chlorine-neutralizing agents being carried over from the washing solution to the chlorine solution. There was no significant difference found in results between establishments employing two-compartment manual dishwashing methods and those employing three-compartment methods. Restaurant operators were more successful in reaching the bacterial standard of 100 or less organisms per utensil surface than were tavern keepers. A lowering of the bacterial standard from 100 to 200 organisms per utensil surface would not greatly increase the percentage meeting the standard. More than half of those that do not meet the present standard have counts in excess of 1000 organisms per utensil surface. The author considers that these represent the hard core of poor operations; if half the group can meet the standard, there is no reason why the other half by employing equal care should not also reach the standard.

Chlorination of Swimming-Pool Water.

E. W. MOOD (*Am. J. Pub. Health*, October, 1953) has investigated methods of adding chlorine to swimming pools to ensure that residual chlorine concentrations will be sufficient to control the bacterial contents. He states that in applying the principles of break-point chlorination to the treatment of swimming-pool water which is recirculated continuously, it was found that the term "break-point" was no longer descriptive of the process. The characteristic "break" could not be demonstrated readily, since ammonia-nitrogen was oxidized continuously as it was washed into the water from the bodies of the swimmers. The term "high-free residual chlorination" has been employed to characterize this newer process of treating swimming-pool water with concentrations of chlorine sufficient to produce and maintain values of free residual chlorine equal to, or greater than, 1·00 part per million. High-free residual chlorination for the disinfection of swimming-pool water presents many new problems. The studies discussed in this paper were not exhaustive but rather exploratory to illustrate the benefits of high-free residual chlorination over marginal chlorination in reducing eye irritation among swimmers and to define areas for future studies. High-free residual chlorination of swimming-pool water reduces the amount of irritation of the eyes of swimmers when compared with marginal chlorination. Optimum conditions of the swimming pool which cause minimal amounts of irritation of the eyes of swimmers are found when the pH of the water is 8·0 to 8·9 and the residual chlorine level is 1·0 to 3·99 parts per million with the principal portion of the fraction as free residual chlorine.

Decontamination of "3-D" Viewers with Ozone.

L. J. WARSHAW (*Am. J. Pub. Health*, December, 1953) investigated public health problems associated with the use of polarizing spectacles by theatre audiences to view three-dimensional films, in relation to the need for effective cleansing of the spectacles before being used by a different person. The author suggests that millions of film-goers must have worn "3-D" viewers which were reissued with little or no effective germicidal treatment; nevertheless, there have been no reports of outbreaks of infection and no apparent increase in the incidence of sporadic cases of skin, scalp or eye infection. However, he considers that the repeated use of "3-D" viewers is at least a theoretical means of spreading infection, and they should be decontaminated before being used again. He suggests the use of ozone and reports that he found that exposure of "3-D" viewers to an ambient ozone concentration of 200 parts per million for twenty minutes completely eliminated a deliberate contamination by pure cultures of *Escherichia coli*, *Micrococcus pyogenes* var. *aureus* (hæmolytic strain) and *Streptococcus pyogenes* (β strain). Exposure to this concentration of ozone for thirty minutes was effective in reducing the deliberate contamination by a variety of fungi to a non-infectious level. Exposure to many times this concentration of ozone had no effect on the optical quality of the polarizing lenses. Exposure to an ozone atmosphere of 200 parts per million for thirty minutes is a satisfactory method of decontaminating or "sanitizing" "3-D" viewers, so that they may be used again without fear of transmitting infection.

Congresses.

THE AUSTRALIAN AND NEW ZEALAND ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE.

THE thirtieth meeting of the Australian and New Zealand Association for the Advancement of Science was held at Canberra from January 13 to 20, 1954, under the presidency of SIR THEODORE RIGG, K.B.E., M.A., D.Sc., F.R.I.C., F.R.S.N.Z., F.N.Z.I.C.

The Sections.

The Sections represented were: A, Astronomy, Mathematics and Physics (including Optometry); B, Chemistry; C, Geology; D, Zoology; E, History and Political Science; F, Anthropology; G, Economics, Statistics and Social Science; H, Engineering and Architecture; I, Medical Science, National Health and Microbiology; J, Education, Psychology and Philosophy; K, Agriculture and Forestry; L, Veterinary Science; M, Botany; N, Physiology and Biochemistry; O, Pharmaceutical Science; P, Geography.

Mueller Memorial Medal.

The Mueller memorial medal for 1954 was awarded to Professor J. A. Prescott, of Adelaide, for research in agriculture. Professor Prescott is Professor of Agricultural Chemistry at the University of Adelaide and Director of the Waite Agricultural Institute.

General Council Meeting.

A meeting of the General Council of the Association was held at the Institute of Anatomy, Canberra, on Wednesday, January 13. There was some discussion of the future relations of the Association to *The Australian Journal of Science*, when the Australian National Research Council should be finally dissolved, after the establishment of the Academy of Science. The position of the Association in relation to the Academy of Science was discussed.

It was resolved, in accordance with a notice of motion given at the General Council meeting of August 8, 1952, that Section I should now be known as the Section of Microbiology, Epidemiology and Preventive Medicine.

Receptions.

On Friday, January 15, Their Excellencies the Governor-General Field Marshal Sir William Slim and Lady Slim entertained senior office-bearers and distinguished visitors at Government House. The presidential reception was held in the late afternoon of Wednesday, January 13, in the grounds of the former residence of the High Commissioner for the United Kingdom at Acton, now part of the Australian National University. Guests were received by Sir Theodore Rigg, Sir Kerr Grant and Lady Kerr Grant, and Professor and Mrs. J. R. A. McMillan. On Tuesday, January 19, the Right Honourable the Prime Minister, Mr. R. G. Menzies, and Dame Pattie Menzies entertained representatives of members of the Association at a late afternoon party at Parliament House.

Inaugural Meeting and Presidential Address.

The inaugural meeting was held in the Albert Hall on the evening of Wednesday, January 13, in the presence of His Excellency the Governor-General, Field Marshal Sir William Slim, G.C.B., G.C.M.G., G.B.E., D.S.O., M.C., D.C.L., LL.D. The senior Vice-President of the Association, Sir Kerr Grant, presented to His Excellency a message of loyalty to Her Majesty the Queen The message had been formulated at a meeting of the General Council of the Congress held on the previous day. Sir William Slim, thanking the delegates for their message, said it would give him great pleasure to convey it to Her Majesty. He welcomed the delegates and wished the Congress success. The newly elected President, SIR THEODORE RIGG, then delivered his presidential address entitled "The Role of Science in the Development of New Zealand Agriculture", in which he gave an outline of the development of agriculture in New Zealand from 1890 to the present day.

Sir Theodore Rigg said that the early period of development, which depended on the felling of forests and clearing of scrubland, had come to an end in 1921; this year marked the end of the pioneering age in New Zealand. From that time on exports of wool and meat had continued to increase. This increase had been associated with the scientific development of areas already under cultivation. Agricultural science had developed enormously since 1921. It had at first met with opposition from farmers, but later had been received with enthusiasm for the benefits accruing from scientific assistance. Sir Theodore Rigg described the value of soil surveys and the discovery of various soil deficiencies. Modern work on "trace element" deficiencies, which had begun in the years 1934 and 1935, had been of great interest and usefulness. There had been an enormous change in the use of fertilizers. Although fertilizer dressing had been very desirable in the early stages of pasture improvement, heavy application on high-yield areas was probably wasteful. The use of fertilizers needed to be controlled by experts. Successful control of pests was referred to. Developments during the past thirty years had demonstrated the value of large staffs of agricultural scientists. It was impossible to assess accurately the monetary value of all this work, but there was no doubt about its real value. During thirty years of the application of science to agriculture the produce of New Zealand had increased enormously. With more workers and larger institutes for agricultural research, the amount and quality of the products of the soil could be improved beyond the present vision, and with them the life and health of the community.

Public Meetings.

On the evening of Tuesday, January 12, a public discussion on "University Education in a Scientific Civilization" was held in the Albert Hall. The Chief Justice, the RIGHT HONOURABLE SIR OWEN DIXON, was in the chair. In his introductory remarks the chairman referred to the large sums spent on education in Australia. He said that modern man had much knowledge at his command, but it was questionable whether wisdom had come with it. He thought that there had been too violent a swing away from the old classical system of education. While it was admittedly absurd to insist that everyone should learn Latin and Greek, it was equally absurd to think that the community would not be the better for a few men who had this approach to a knowledge and understanding of the past. He emphasized the importance of motive in education and in the choice of one's work in life. The pursuit of learning for its own sake brought wisdom in its train, no matter what branch of human knowledge was being studied. What made all the difference was not so much what a man did as why he chose to do it.

DR. I. CLUNIES ROSS, Chairman of the Commonwealth Scientific and Industrial Research Organization, said that it was extremely difficult to escape the pressure of the specialized—and therefore limited—training which all university graduates must now have. The demands of science were such that the interests of trainees must be narrowed because of the increasing amount of human knowledge, and the thoroughness with which it must be learnt. The demands of society for trained scientists and technologists had increased enormously. The remorseless pressure of civilization was such that universities were no longer universities in the old sense of the word. The emphasis was more and more on technical training, especially in the faculties of medicine and engineering. He believed that reform was necessary, but it was difficult to say of what kind and how it could be brought about. As for graduates being capable of considering any problem that arose, he knew how, in his own case, it was absolutely impossible, because he had been forced to concentrate on a particular field of knowledge. Under those conditions, the consequences for the future would be grave. There would be fewer and fewer who could actually confront problems of society and find a solution on a question of principle or values. The changes which science had brought about were relatively superficial. Underlying the surface of the scientific world were the same individuals, and the solutions to many of the world's problems must take account of the behaviour of human beings. Rosy hopes for solutions to those problems had proved false again and again, because there was no approach with sound wisdom underlying it.

Universities must be changed, but how? If they could clarify and redefine in intelligible terms what the functions and purposes of Australian universities were, they could face the next step of attempting to determine how those functions could be discharged. Neither governments nor any other bodies had ever devoted sufficient attention to the broad purposes of education.

PROFESSOR J. P. BAXTER, of the New South Wales University of Technology, also discussed the place of a university in modern civilization. He said that the functions of a university were twofold: first, the function of teaching, and second, the pursuit of knowledge for its own sake. Both were necessary. The former meant the training of men to control and operate present-day scientific civilization. It should include

post-graduate as well as undergraduate teaching. Australia could make no better investment than to contribute £1,000,000 a year from taxation to provide post-graduate training. Such an action would pay great dividends to the country. As it was, the influence of universities on the men who actually operated and controlled civilization had steadily declined. The universities believed that they had something to give to the people outside them. A broader contact between the universities and the community would benefit both. Professor Baxter asked in what esteem university professors were held by the people of Australia as a whole. He said that there was a section of the community that held those who worked in universities in some degree of contempt. That feeling was very pronounced in Australia, perhaps more than in any other country. It could be found in government officials, in the public service and in industry. It was seldom aimed at individuals, but it was directed at professors as a group. That might seem a trivial matter, but it was indicative of a situation from which both the communities and the universities suffered. It maintained a gulf between the universities which must be bridged. A solution might be sought in liaison panels representing both universities and industry, and in post-graduate courses and extension lectures. Proceeding to the second function of a university —namely, research—Professor Baxter said that it might be fostered by grants for the training of recent graduates in research methods.

An animated discussion followed, during which one speaker, after describing the many tragic and comic aspects of present-day civilization, asked on what grounds it should be called a "scientific" civilization. At the conclusion of the discussion, PROFESSOR A. P. ELKIN (Sydney) put the following motion:

> That the pace of scientific development and the rapid growth of population have seriously affected the function and structure of tertiary education in Australia; that the time has come for a thorough investigation of our universities, which are labouring under many handicaps; and that the Australian National Research Council be asked to explore the possibility of appropriate action.

The motion was carried almost unanimously and was received with acclamation by the large gathering present at the meeting.

Public Lectures.

On the evening of Thursday, January 14, a public lecture on "Australian Exploration in Antarctica" was given by SIR DOUGLAS MAWSON in the Albert Hall.

On Sunday evening, January 17, PROFESSOR N. E. ODELL gave an illustrated lecture entitled "The Himalayas".

On the evening of Tuesday, January 19, a lecture entitled "Recent Developments in Astronomy" was given by PROFESSOR OTTO STRUVE, Director of the Department of Astronomy at the University of California and President of the International Astronomical Union.

The Masson Lecture.

The Masson Lecture on "Research, the Basis of a Major National Project" was given by DR. R. S. ANDREWS, Chairman of the Victorian Gas and Fuel Corporation. Dr. Andrews described the manufacture of town gas and synthetic liquid fuel by pressure gasification of brown coal from the vast deposits in the Latrobe Valley, Victoria. He said that this was a giant revolutionary project; it was now well under way and would be completed in 1956. It must be evident to all that far greater development of coal resources was necessary if Australians were to maintain their living standards. Australia must mine at least 70,000,000 tons of coal a year (four times her present output) if she was to have a population of 20,000,000. At present 77% of Australia's coal requirements came from New South Wales, and all were coking coals. The large deposits of non-coking coals in other States had not been seriously exploited in relation to the overall development of the Commonwealth. Victoria possessed in brown coal the equivalent to 5000 million tons of black coal. It thus became a necessity for her to utilize brown coal in industry, in traction, for power generation and for town gas production. Before such a programme could be implemented it was essential to have a precise knowledge of the fundamental chemical and physical properties of brown coal in relation to its use. Dr. Andrews showed how the Latrobe project fitted into the future development of the Commonwealth, and how each step had been accompanied by fundamental and applied research.

Visits to Scientific Institutes.

On Wednesday, January 13, members of the Congress inspected the laboratories of and exhibits by the Divisions of Entomology and Plant Industry and the Section of Land Research and Regional Survey of the Commonwealth Scientific and Industrial Research Organization. On Thursday, January 14, members were invited to inspect the laboratories of the Research School of Physical Sciences, Australian National University. On Saturday, January 16, members of Section I were invited to inspect the laboratories of the John Curtin School of Medical Research, Australian National University. Members of Section A with many members of other Sections visited the Commonwealth Observatory, Mount Stromlo, on the afternoon of Saturday, January 16.

Church Services.

On Sunday, January 17, a special service for delegates was held at St. John's Church of England, where the preacher was the Right Reverend E. H. Burgmann, Bishop of Canberra and Goulburn. The lessons were read by Professor M. L. E. Oliphant and by Sir Theodore Rigg. A special service for delegates was held at St Andrew's Presbyterian Church. At a special Mass at St. Christopher's Roman Catholic Church, the preacher was the Most Reverend Eris O'Brien, Roman Catholic Archbishop of Canberra and Goulburn.

Symposium.

On the evening of Monday, January 18, a symposium on the history of science in Australia was held under the chairmanship of SIR DAVID RIVETT. The speakers were PROFESSOR A. A. ABBIE (Adelaide) and DR. R. S. BURDON (Adelaide).

A. A. Abbie, discussing the history of biology in Australia, said that recorded biological discovery in Australia had begun with Pelsart in 1627. He had been followed by Volckersen and by Dampier, who described some animals, plants and aborigines in 1688 and 1699. Cook's voyage in 1769-1770 was occasioned by a number of unlikely coincidences, exploration of the east coast of Australia was almost accidental, and only the presence of Banks and his party lent any biological significance to the venture. Banks continued to encourage Australian development and exploration until his death in 1820. The first half of the nineteenth century saw tremendous advances in biological knowledge and ideas and culminated in Darwin's theory of evolution. But the Australian evidence, although it should have supported the theory strongly, was ignored in the early stages and came into its own only later. The latter half of the last century was distinguished by vigorous exploration and more systematic biological work, especially in the field of bioeconomics. At the same time, most of the existing learned bodies were established. The twentieth century had continued the trend of the nineteenth, but with increasing regard for conservation and restitution in place of exploitation and destruction. There had also been more determined and systematic warfare upon pests and parasites. In both these latter activities the Commonwealth Scientific and Industrial Research Organization had played a very important part. Finally, there was evidence of a growing maturity in dealings with people of other countries. That improvement must be accelerated if Australia was to survive.

The Ecology of the Staphylococci.[1]

In her presidential address to Section I, PHYLLIS M. ROUNTREE (Sydney) said that the choice of a specific rather than a general topic as her subject required perhaps some justification. However, if some general conclusions could be drawn from a study of the behaviour of one particular group of biological entities, such a study might have some value. It was not so long since many orthodox geneticists had denied the suitability of bacteria and viruses as material for genetic study. Old attitudes died hard, but at the recent International Congress of Genetics at least 20 papers had been read on various aspects of microbiological genetics. Ecological studies had up to the present been made preponderantly with the sexual organisms that were the classical material of genetics; but, as Burnet had ably shown, bacterial ecology could be examined in a similar way.

The distribution of staphylococci was obviously correlated with intrinsic characters that determined their ability to occupy certain ecological niches; but since they were parasites or commensals of higher animals, the nature of the reactions of their hosts to their presence was also important in determining their distribution. Their habitat in man and other mammals would therefore be discussed, together with the role of intraspecific competition in determining the occupation of individual "micro-environments", the recent emergence of antibiotic-resistant races and their establishment in particular environments—a phenomenon

[1] This paper will be published in full in the proceedings of the Congress.

which provided a striking illustration of the process of what Dobzhansky had called "micro-evolution". The chief principle revealed by those characters of the staphylococci was the necessity of a species to possess genetic variability. Their main habitat was the animal body, and they were therefore found in all materials that were in contact with animals. Since there was a wealth of observations on the pathogenic staphylococci, and very few on other species, the pathogenic coagulase-positive species *Staphylococcus aureus* Rosenbach emend. would be discussed in what followed.

Staphylococcus Aureus in Man and Animals.

Dr. Rountree said that the recent increase in knowledge of *Staph. aureus* was largely due to the introduction of phage typing, which had become a valuable tool in epidemiological studies. In man the normal habitat of *Staph. aureus* was the anterior nares. Numerous surveys had shown that the average percentage of nasal carriers was usually about 50. Under certain conditions the rate might rise to 80%. Furthermore, 90% of the infants born in hospital became profuse carriers within their first week of life. The nasal carrier state in man was not, however, a static one, but was continually changing. A small proportion of people appeared to be non-carriers; the reason for that was not known. There was little information about the carrier state in other animals. In the course of a study of tick pyæmia in lambs, Foggie had found pathogenic staphylococci in the mouth, nose and vagina of ewes in both tick-infested and clean flocks. Dr. Rountree described a recent investigation of the nasal flora of some species of animals in Sydney; the only species so far examined that had significant carrier rates were monkeys. Despite the occurrence of pathogenic staphylococci in other mammals, it was *Homo sapiens* who was their host *par excellence*. Man's association with the staphylococci went back into antiquity. From long association a nicely adjusted balance had been achieved, but from time to time the stage of toleration broke down in individuals. The advent of the antibiotic era had been hailed as revolutionizing the treatment of staphylococcal infections; but such prophecies had been unduly optimistic and had taken no account of the adaptability of the staphylococcus. Dr. Rountree discussed the various lesions caused in man, and said that the host-parasite relationship in staphylococcal immunity presented many unsolved problems. A study of the nasal staphylococci of man had revealed the interesting point that it was rare for more than one phage type to be isolated from a given host at one time. This might be an instance of intraspecific competition. A strain well established in the nose remained in possession of its ecological niche, even though the host was exposed to an environment heavily saturated with strains of a different type.

Evolutionary Changes in the Staphylococci.

Dr. Rountree went on to say that the emergence of antibiotic strains of the species *Staph. aureus* had provided a striking example of the selective effect of changes in environment and of a startlingly rapid process of "micro-evolution". She discussed this process in detail, from the point of view of genetics. She went on to discuss streptomycin resistance and genetic instability, and also mutation resistance to other antibiotics. Staphylococci in hospital environments had been able by genetic change to adapt themselves to the presence of antibiotics with remarkable success. That process was still continuing and could not be regarded with any equanimity. The use of any newly discovered antibiotics should therefore be rigidly controlled so that they might be restricted to the treatment of patients with serious infection with resistant strains, for which the older autibiotics were of no value. When the new substances were used the patient should be kept in strict isolation, and if resistant mutants did appear, every precaution should be taken to prevent their dissemination in the hospital. In conclusion, Dr. Rountree said that man was an inveterate meddler with his environment. Nearly all the ecological changes occurring within living memory had been due to his activities. His meddling had now included pharmacological attacks on his own microbial flora; and in certain cases the results had been unforeseen. For example, the oral administration of aureomycin, chloramphenicol or terramycin had resulted in the destruction of the normal intestinal flora, which had been followed by the development of vitamin deficiencies and also of a flourishing fungal flora. On the other hand, some of these pharmacological attacks had been outstandingly successful; an instance was the fact that hæmolytic streptococci had almost disappeared as a cause of hospital cross-infection. The hæmolytic streptococcus had been unable to adapt itself to a changing environment. The more mutable staphylococcus had scored an outstanding genetic victory, and the recent natural history of these two microorganisms illustrated very well the necessity of biological adaptability if a species was to survive the challenge of a changing environment.

Dietetic and Nutritional Factors in Relation to the Prevention of Dental Caries.

H. R. SULLIVAN and N. E. GOLDSWORTHY (Sydney) showed a film illustrating food habits which ensured the maintenance of adequate nutrition and of sound teeth. It was stated that the children in the film had been examined by members of the staff of the Institute of Dental Research at more or less regular intervals since the beginning of 1947. They lived as a community in a large home with comfortable anexes in a fertile country area at Bowral, New South Wales. Physically they were much the same as children of the same age living as members of the general population. Dental examination had shown that their teeth were remarkably free from carious lesions. Indeed, the average number of decayed and filled teeth per child at the end of February, 1952, was only 0·58, whereas children in the same age groups in the general population rarely passed their eight birthday without suffering from caries, and had on an average ten to twenty times more decayed, missing or filled teeth. The outstanding difference between the children in the film and groups living in the population at large was in the nature of the diet. The speakers emphasized the necessity of defining the terms diet and nutrition. Diet meant the food taken regularly by an individual, whereas nutrition was the sum of the processes concerned in the growth, maintenance and repair of the living body as a whole or of its component parts. The types of food eaten by the Bowral group were: vegetables, raw and cooked; salads; fruits, fresh and dried; wholemeal cereals and wholemeal bread; milk, butter and concentrates of vitamins. The intake of sufficient protein was assured by the free use of milk, cheese, soya bean and egg. In contrast, the types of food eaten by children in the general population included white bread and jam, cakes, pastries, sweets and ice cream, sometimes at the expense of the "protective" foods, but more often in addition to them.

There was ample evidence to justify the claim that there was a close relationship between the ingestion of large quantities of refined carbohydrates and the prevalence of dental caries: evidence gleaned from studies of native peoples, from populations subjected to wartime rationing, and from animal experimentation. The general assumption based on such observations was that, though a diet might be nutritionally adequate, its carbohydrate moiety (in particular) had been factory processed—concentrated, deprived of all or most of its fibrous structure and often left devoid of its original content of inorganic salts and vitamins. This "refined carbohydrate" provided, in a form which would readily cling to and around the teeth, ample substrate from which acidogenic bacteria might form lactic acid, resulting in a dissolution of the teeth. In the absence of white bread, prepared breakfast cereals, biscuits, cakes, sweets and sugar from the diets of the Bowral group of children, as well as the absence of any other likely factor such as fluoridated water, it seemed reasonable to assume that their freedom from caries was due to this absence of refined carbohydrate from their diet.

It was held that the most desirable state of freedom from caries presumably could be attained by any person who was prepared to adjust his dietetic habits or those of persons in his charge so that the Calorie intake was maintained by eating the products of wholemeal flour and other cereals and carbohydrates in their natural state. He must also see to it that the carbohydrate intake satisfied natural nutritional requirements, but did not greatly exceed them. Under the conditions of western civilization the use of refined carbohydrates, including white flour products, had become an accepted, almost basic, part of the dietary pattern. A sustained effort would be necessary if they were to establish a new dietary pattern. However, to change the food habits of communities or even of individuals was extremely difficult. The speakers proceeded to discuss this problem of changing food habits in greater detail.

When the problem of changing food habits in order to bring about a reduction in dental caries was considered, it quickly became evident that the very nature of the disease made the acceptance of dietary changes difficult. The aches and pains of the disease might be temporarily distressing, but eventually they disappeared, and even the loss of all the teeth seemed to be looked on by some as but a slight physical handicap. Then, too, the relationship between the diet and the disease was not as obvious as in such conditions as diabetes and allergy. Also, they had to face the fact that there were numerous prejudices which made dietary

changes extremely difficult. Foremost was the prejudice arising from ignorance. Any suggested changes were looked on by many people as not merely an interference but a deprivation. One of the greatest difficulties was to persuade people that a change in the nature of its carbohydrate intake did not mean that a child was being deprived of some desirable (implying both tasty and necessary) commodity. Another prejudice arose from the existence of different social strata. That fact had been operative throughout the ages. Thus it was the upper classes of the Egyptians, who because of their position ate a diet rich in carbohydrate, that developed dental caries, whereas the slave population remained practically free. Another prejudice related to this sprang from the existence of people living within a community, yet retaining a cultural background which had imposed a rigid dietary pattern on them. This was not seen so much in Australia as in the United States of America; but with the continual inflow of New Australians it could become important. Linked with the prejudices arising from ignorance, from social habits and from cultural background were certain difficulties to which, at present, there appeared to be no solution. As the living standards of the world changed there had arisen a demand for greater supplies of foodstuffs to ward off famines and to establish a reasonable intake of nutrients. It seemed to have become necessary (with the present limited knowledge) to prepare foodstuffs in refined concentrated forms—such as white flour and sugar—in order that they might be stored for considerable periods of time. Even in a limited community the cost of maintaining a diet made up of a great proportion of vegetables, fruit, eggs and the more expensive types of cereal products might prove to be beyond the financial ability of many.

The last difficulty was the innate love of sweetness which most people seemed to possess. That attribute, linked with the fact that dental caries was not directly a killing disease, was probably the key to many of the difficulties experienced in trying to prevent caries.

What suggestions could be made as to how to effect changes in food habits? First, the relationship between nutrition and caries should be determined and then one could estimate to what level it would be desirable to lower the intake of refined carbohydrate in order to prevent extensive caries. Much had been written about the relationship between nutrition and dental caries. Mellanby's experimental evidence clearly indicated that impaired nutrition could interfere with the formation of normal teeth. However, there was no unanimity about the association between imperfect structure and caries. Also it had been observed that a nutritionally deficient diet was compatible with a high resistance to dental caries. However, the compatibility of such diets with freedom from caries was almost certainly due to the chemical and physical nature of the diet rather than to its nutritional inadequacy, and to the fact that populations receiving suboptimal diets were probably restricted to two or three meals a day, with no between-meal snacks. While nutrition could be related to structure, it was not necessarily related to freedom from or susceptibility to caries. Merely from basic principles the speakers considered that good nutrition was advisable in order to have good tooth structure and good bodily health.

Turning to a consideration of an acceptable level of refined carbohydrate intake, the speakers pointed out that it was well known that wartime rationing of sugar and sweets had been sufficient to bring about an appreciable decrease in the amount of caries in many European countries. From observations made in Scandinavia it appeared that eating between meals might be quite a big factor in caries, especially because so-called snacks generally consisted of refined carbohydrates. The problem was how, without upsetting their nutritional status, to change the diet (both the food pattern and the feeding habits) of the population in order to restrict the intake of refined carbohydrate and to ensure its ingestion only during normal meals. In a country like Australia or New Zealand, where there was a plentiful supply of foodstuffs, the problem would be all the harder. In these countries "good eating" usually became a matter rather of seeking some pleasant variation in dishes than of choosing foodstuffs for their nutritional and dietetic value. The main tool would appear to be education through persuasion. The speakers quoted Galston, who had pointed out the fallacy of believing that facts starkly presented would prove to be compelling arguments, and had outlined the more scientific and realistic method of persuading people to change their habits by appealing to their innate motives. All health education rested upon the assumption that people prized health and that they were eager to be healthy. According to Galston, the data of dynamic psychology showed that this assumption was invalid. No such instinct, drive or motivation was known to dynamic psychology. The individual wanted to eat, to move, to rest, to serve his body's needs. Grown older, human beings wanted love, sexuality, marriage, a home and children. To achieve fulfilment the individual needed to be healthy. According to Galston the true goals of the individual must be used for health education if it was to be successful. Sherman had believed that people could be persuaded to change their food habits and had quoted figures indicating increased per-capita consumption of milk, vegetables and citrus fruits and tomatoes in the United States of America in 1945 as compared with 1915. It seemed to the speakers that in communities such as Australia, changes in dietary habits could best (perhaps only) be effected by a combination of discipline and persuasion in different proportions according to the mental and intellectual status of those towards whom efforts were being directed. Education in ways of preventing caries was obviously not the responsibility of the dentist alone. Such education must begin with the medical practitioner in the prenatal clinics, must be carried on in baby health centres, in kindergartens and schools, and should be continued during the adolescent period. Perhaps the most successful application was that made during pregnancy and the nursing period, when the mother would readily accept variations in her usual dietary pattern if they were likely to benefit her child. If similar advice along those lines could be given to mothers at child health centres, the lessons begun earlier could be reinforced to a point where the motivation pattern became firmly established.

Animal Diseases Communicable to Man.

A joint meeting of Sections I and L took the form of a symposium on "Animal Diseases Communicable to Man".

Introduction: Medical Aspects.

I. M. MACKERRAS (Brisbane) gave a brief review of the main groups of infections which reached man directly or indirectly from animal hosts. Meyer had listed 75 diseases of man which had animal reservoirs. Wright had recorded no less than 116 species of parasitic worms which included man at least occasionally among their hosts. Dr. Mackerras showed a series of slides, giving classified lists of the many infections in those groups which had occurred in Australia. The list of helminthic infections comprised six varieties of nematodes, seven of cestodes and two of trematodes. Passing to protozoal and fungal infections, he said that that group was of doubtful validity in Australia where hæmoflagellates infecting man were absent, and were not likely to be acquired, because the vectors were either absent or rare. Thus, apart from some ringworms, of which the animal reservoir was undoubted, there were left Toxoplasma and Sarcocystis[1] among the protozoa and several systemic mycoses among the fungi. In these the animals might not be reservoirs, but simply victims, like man, of infections derived from their environment. The next group, that of bacterial infections, included bovine tuberculosis, erysipeloid, listeriosis, leptospirosis, the rat-bite fevers, plague, melioidosis, brucellosis and salmonellosis. Apart from epidemic typhus, which had been brought to Australia in the early days of the settlement but had not become established, four types of rickettsial infections were known in the country. These were typical animal reservoir infections, in that man was an accidental intruder into a cycle with which he had normally nothing to do. Coming to viral infections, Dr. Mackerras gave the following list: psittacosis, Murray Valley encephalitis, contagious ecthyma, and cat-scratch disease.

Dr. Mackerras proceeded to discuss some problems in ecology, raised by the study of that group of diseases. He said that the list he had given seemed a formidable one, but the majority of animal reservoir diseases were uncommon, and many were no more than clinical and zoological curiosities. Sporadic, scattered, apparently unrelated appearance in man was, in fact, a normal characteristic of that group. The second characteristic was related to the first; it was that in the majority of these infections man formed no part of the ecological niche which the parasite normally occupied. That fact had one important practical consequence—namely, that controlling those infections in man in no way influenced the effective parasite population; to do that they must be controlled in the animal host, and that was often a much more difficult task. A third characteristic of that group of infections was that the reaction provoked by the parasite often differed greatly in its principal and casual hosts. Dr. Mackerras went on to

[1] Dr. Mackerras said that Spindler (1947) believed that Sarcocystis was a fungus.

discuss the phenomenon of adaptation. He said that the practical consequence of it was that one looked for the disease in patients, but for the infection in the reservoirs, though there were some important exceptions to that rule. The frequency of these infections in man depended on a number of factors, which were discussed under the following five headings: the spread within the human population, the dosage of infection, the pathways of infection, the portal of entry, and the association between hosts. Lastly, Dr. Mackerras spoke of future research in that field. He said that at present much could be done to prevent or ameliorate the animal reservoir infections. Veterinarians could control infection in domestic animals; meat and food inspectors could prevent it from reaching the human consumer. Vermin control still left much to be desired, though substantial progress had been made in that field. The occupational bias of some infections had been recognized. It was impossible to control bush rodents or Trombiculine mites, but McCulloch had developed the use of dibutyl phthallate to act as a barrier between the mite and its casual human host—and now chloramphenicol had robbed scrub typhus of most of its terrors. As to the future, it was for the microbiologist to discover whether there were any still unrecognized infections that attacked man. Next came the search for reservoirs and vectors, and there the zoologist began to appear as an essential member of the team. Habitat studies must follow, and there the task passed from the microbiologists and zoologists to the field ecologist. Too little work of that kind had been done in Australia. Dr. Mackerras said that he had been very impressed with the effect that small changes in the environment produced by even sparse settlement had made on many native animals; they had vanished away from places where they had once been common. He thought, therefore, that equally small changes, provided they were of the right kind, might drive away some of the reservoir rodents and bandicoots from the places where they were not wanted. That could be revealed only by careful, detailed habitat studies. It was certainly worth trying, and the general knowledge obtained would in any case not be wasted. He had a strong aversion from the extermination of any of the native fauna—too much of it was disappearing already—but a safe balance between control and extermination could be achieved only through such studies as he had indicated. Last should come population studies which would carry the ecological investigations to their logical conclusions. In the words of Elton: "Animal population dynamics . . . promises to become the fundamental science upon which pest control and protection from animal diseases would be based."

Introduction: Animal and Veterinary Aspects.

L. B. BULL (Melbourne) discussed the problem of parasitism and the susceptibility of species to particular parasites, including helminths, arthropods, protozoa, bacteria and viruses. The term "anthropozoonoses" had been coined to describe diseases common to man and animals. Some of the infestations or infections contracted by man from animals were of definite biological interest, but were of little or no significance in public health or animal health considerations. On the other hand, some of the diseases contracted by man from domestic and wild animals were of great public health and animal health importance. Included among these were leptospirosis, "Q" fever, brucellosis, bovine tuberculosis and psittacosis. It was regrettable that there was in Australia no provision for public health work in veterinary diseases in regard both to those that were communicable to man and to those that were not. Dr. Bull went on to describe the epidemiology, pathogenesis and control of various diseases common to man and animals.

Psittacosis.

J. A. R. MILES (Adelaide) reviewed the history of psittacosis in Australia. He said that psittacosis-ornithosis viruses infected a wide range of birds in Australia. Infection had been recorded in 16 species of psittacines and in at least six species of birds from other orders. Infection was widespread in the wild birds, but a much higher percentage of aviary and of domestic birds had been found to be infected. Despite a large amount of avian infection few human cases had been recorded; the notifications, at least in South Australia, represented not more than half the proven cases, but even when allowance was made for this, the number of cases was small. The high mortality recorded in Australia was probably due to the failure to notify mild cases. The Australian strains of virus had all been found to have a low pathogenicity for mice, especially by the intraperitoneal route, and it was suggested that strains of that type had a low infectivity and pathogenicity for man. Psittacosis-ornithosis viruses had a remarkable capacity for establishing chronic infection with little ill health, both in their ordinary hosts and in new hosts such as mice and men; but in new hosts they seemed to have difficulty in spreading from case to case. However, small epidemics with man-to-man spread did arise from time to time, and a case was on record in which a man had been intermittently excreting virus in his sputum for ten years and was still continuing to do so. At present the number of new cases of psittacosis occurring in Australia was small, so that the disease constituted only a minor public health problem. However, quite a small variation in the character of the virus might give it a much greater significance for man.

Leptospirosis.

A. K. SUTHERLAND (Queensland) discussed the occurrence of leptospirosis in animals. He said that it occurred throughout the dairying districts of Queensland, except the Atherton Tableland, and also in Victoria, New South Wales and Western Australia. It was usually confined to the coastal belt. A spread to the inland districts, when it occurred, was associated with the transport of herds of pigs. As to treatment, he believed that there was a feeling in human medicine that antibiotics were of little use. Work in New Zealand had shown that penicillin was of little value in the treatment of infected animals, streptomycin was better, and aureomycin was the best drug tried so far. The epidemiology had not yet been explored. It was known that symptomless infections occurred in cows and calves, that cows occasionally were carriers of the disease, and that pigs could be heavy carriers. The speaker referred to the ability of the organism to survive in mud and water and to enter cuts and abrasions. As to the control of the spread of the disease and its conveyance to human beings, they were largely a question of agricultural architecture and engineering. It was surprising what a number of men worked with their feet bare in places where leptospirosis was known to occur. The high incidence of carrier infection in pigs was well known. Where it was high, the sows produced dead or weak litters; it had been shown that that was not due to brucellosis or other common causes of that condition, and each herd in which it had occurred was heavily infected with *Leptospira mitis* or *Leptospira pomona*.

E. H. DERRICK (Brisbane) read a paper entitled "Epidemiological Observations on Leptospirosis in North Queensland". He said that leptospirosis was endemic in a narrow, well-watered, coastal belt of North Queensland, extending from Ingham to Cooktown. During the two years from 1951 to 1953, 152 cases from that area had been studied. They had been caused by eleven types of Leptospira, the commonest being *L. australis* B., *L. australis* A., and "Kromastos". Geographical analysis had shown a patchy distribution. Cases tended to arise in the vicinity of rivers and creeks and were much commoner in some areas than in others. Very few cases occurred on the tablelands. Among other areas, cases had occurred near Mossman, and it appeared that leptospirosis was a major constituent of "Mossman fever". Wherever leptospirosis occurred there was likely to be a profusion of leptospiral types. Cases of leptospirosis in Johnstone had shown a striking correlation with the rainfall occurring eight to eighteen days before the onset, the commonest time being twelve days. As the incubation period of leptospirosis, as found by Schuffner, was four to nineteen days with an average of ten days, there was a rapid development after rain of conditions favouring human infection. In the exceptional cases of leptospirosis occurring in dry weather, a history of contact with water, from spear-fishing, swimming or cleaning drains, could sometimes be elicited. Dr. Derrick said that the literature contained reports of infection by direct contact; but in his cases contact with infected water seemed the most likely mode of infection. He showed photographs of typical country in the canefield areas. The combination of mountain, river and canefield favoured the occurrence of leptospirosis. He referred to the practice as a preventive measure of burning the leaves of the canestalks, thus destroying the leptospiræ and driving out the rats.

Nocardiosis in Dogs.

K. G. JOHNSTON (Sydney) said that granulomata were important in canine pathology because they might be due to infective and transmissible agents. The organisms concerned in the ætiology of mammalian granulomata were the Mycobacteria, the Actinomycetes (including the Nocardias), and the Fungi Imperfecti. The commonest infectious granuloma of the dog was tuberculosis. More recently nocardiosis had been encountered. This infection had occurred in human beings in Australia. The outstanding features were chronic sinuses of the subcutis and a primary pseudo-tuberculosis, often with dissemination. Illustrations

depicting the lesions in two young dogs with this disease were shown. Transmission from animals to man had not been reported. However, infected dogs constituted a reservoir from which other mammalian hosts might possibly become infected.

Brucellosis.

In a symposium on brucellosis, T. S. GREGORY (Melbourne) described the veterinary aspects of the disease, M. M. WILSON (Melbourne) described the use of the antiglobulin test in the diagnosis of brucellosis, and W. J. STEVENSON read a paper entitled "A Comparison between the Antiglobulin Test and the Skin Test in Brucella Infection". He recorded the use of these two tests in an investigation of Brucella infection in abattoir workers. He said that the investigation had been initiated by the Fairfield Epidemiological Unit in collaboration with the State Health Department, Melbourne. "Brucellergen", a protein nucleate fraction of Brucella cells, was the skin test antigen employed. An intradermal injection of 0·1 mil was used and the reactions were read forty-eight hours later. Blood samples had been obtained from the 71 subjects of the study immediately prior to the performance of the intradermal tests. Tests for Brucella antibodies in the serum had consisted of the direct agglutination test and a modified Coombs antiglobulin test, as adapted by Wilson and Merrifield. (The terms "complete" and "incomplete" antibody were used for convenience and referred to antibody detected by the direct method and by the Coombs technique respectively.) In this group of 71 abattoir workers there was close correlation between the existence of sensitivity to "Brucellergen" and the presence of circulating "incomplete" antibody. Of 40 men with positive skin reactions, the sera of 37 had shown titres of 1:5 or more in the Coombs test. Conversely, of the 44 men whose sera had shown titres of "incomplete" antibody of 1:5 or 1:10, about half had been sensitive (five of twelve); of those whose sera had given negative results at 1:5 dilution, three of twenty-seven were sensitive to "Brucellergen". The specifity of "Brucellergen" and the modified Coombs test appeared to be mutually confirmatory. Such discrepancies as had been noted had been confined to borderline findings in either test, weak skin reactions or low "incomplete" antibody titres (less than 1:20). The systemic and complicating local reactions which had been observed in some subjects who had had positive skin reactions had caused some concern. The possibility of such reactions occurring in a hypersensitive group such as abattoir workers tended to militate against the use of "Brucellergen" as an epidemiological tool.

Duration of Infection with Coxiella Burneti in Man and Animal.

E. H. DERRICK (Brisbane) contributed a series of observations on the incubation period in human infections with *Coxiella burneti*, the period during which *C. burneti* might be isolated from the urine, and the duration of the presence of agglutinins and complement-fixing antibodies in the serum. A series of similar observations in the guinea-pig was also recorded.

The Natural History of Murray Valley Encephalitis.

S. G. ANDERSON (Melbourne) said that Murray Valley encephalitis was believed to be identical with Australian X disease. Since 1913 there had been five outbreaks of the disease among humans in eastern Australia. The morbidity was low, but among clinical cases the mortality had averaged 65% over the five recorded epidemics. The causal virus was a member of the arthropod-borne encephalitides, closely related to, but distinct from, Japanese B encephalitis. The infection, at least during 1951, had been widespread among native and domestic fauna, though little or no disease had been detected in animals. Although the five epidemics had been recorded in the Murray Valley and eastern Queensland, the virus did not "overwinter" in the Murray Valley. The endemic foci of the virus were probably located in the general region of the Gulf of Carpentaria, where the virus persisted in a series of avian-culicine cycles. The periodic epidemic episodes in eastern Queensland and the Murray Valley were due to the spread of the virus to the east and south from the endemic area. The spread was determined by the occurrence of grossly excessive spring rainfall in eastern Australia. That ecological situation might have a more general application to the overseas members of the arthropod-borne encephalitides.

Mosquitoes as Experimental Vectors of Murray Valley Encephalitis.

D. M. MCLEAN (Melbourne) said that during the period from January to April, 1951, the virus of Murray Valley encephalitis (M.V.E.) had produced 45 clinical cases and many subclinical infections in the Murray Valley and further northwards. The epidemiological findings had suggested that the disease was mosquito-borne. Experiments had shown that M.V.E. virus underwent a biological cycle in the mosquito, and that several species of culicine mosquito might act as vectors of M.V.E.

Section I: Nutrition.[1]

Nutrition in Pregnancy.

J. WOODHILL (New South Wales) discussed the influence of maternal diet in pregnancy. She said that there were women living in the city of Sydney who were eating diets sufficiently low in nutritive value to affect their chances of having a completely successful pregnancy followed by a period of satisfactory lactation. A study of 186 pregnant women who were racially homogeneous had shown that there was an important relationship between pre-natal diet and reproductive performance. Previous to their pregnancies 15% of the group had been eating diets which, in respect of most of the nine nutrients calculated, contained less than 50% of the recommended daily dietary allowances of the National Health Research Council of the United States of America. In the latter half of pregnancy the number of women with deficient diets increased from 15% to 20%. A statistically significant relationship had been demonstrated between poor maternal diets and the incidence of toxæmia and prematurity, and between good maternal diets and the duration of lactation. There was a significant association between primiparity and toxæmia; but the *primiparæ* had not been consuming diets inferior to those of the *multiparæ*. Preconceptional obesity was related to the occurrence of toxæmia and the early loss of milk. The mothers in the study who had been receiving good diets had given birth to babies who were heavier and larger than those born to mothers on poor diets. In a prosperous community such as was represented by the subjects of the study, the problem still remained of finding those particular individuals who needed special nutritional advice during pregnancy. The importance of good nutritional status at the beginning of pregnancy had been demonstrated, and the improvement of the diets of Australian mothers should start before conception had taken place. One goal in the public health programme to promote maternal and child health should be the education of women in good dietary practices in preparation for motherhood.

Developments in Infant Feeding.

R. R. GIBSON (New South Wales) discussed the field of nutrition in infancy from the point of view of a physician whose main interest was in gastro-intestinal medicine. The history of a case was quoted to illustrate the importance of early feeding situations in the development of gastro-intestinal disorders. The importance of the emotional environment of mother and child as a feeding unit was stressed. The application of those principles to hospital practice was described.

Nutrition in Old Age.

C. TURNER (Melbourne) said that much attention had been drawn recently to the increase in the number of elderly persons in the community. The percentage of the population aged over sixty-five years had doubled in the last thirty years. Since its inception seven years earlier the Clinical Research Unit of the Walter and Eliza Hall Institute and the Royal Melbourne Hospital had been aware of the increasing importance of the problem and of its nutritional and socio-economic implications. Recently 70 patients aged over sixty-five years had been selected for special study by a physician, a dietitian and an almoner; it had been shown that the years of useful activity of the older members of the community could be prolonged. Good health and happiness in old age depended on a triad of factors—physical health, social status and nutritional status. Those three factors should so operate that the activity, both physical and mental, of elderly people gradually diminished rather than ceased suddenly at an arbitrary retiring age. Much could be achieved by the cooperation of the physician, the almoner and the dietitian to assist old people to live active, happy and useful lives, often to the end of their days.

Miss Turner said that the following factors were desirable in the diet of old age: a gradually decreasing caloric intake, but a steady inflow of protein; a low intake of fats, since fats could hasten the development of arteriosclerosis; a

[1] During two of the five days on which papers were read, Section I split up into two series of concurrent sessions, one of which dealth with microbiology, the other with nutrition.

high level of mineral and vitamin requirements; bland and easily masticated food with considerable bulk but no roughage. Obesity had detrimental effects in old age, and obese patients soon felt the benefit of a low Calorie diet. Some women, on the other hand, had been expending energy far in excess of their caloric intake by keeping house or helping daughters with their families. The health of those women had improved remarkably when they had begun to eat more and to improve their diets. Excessive consumption of alcohol was sometimes a factor in malnutrition, but it was not a common thing. The younger generation was growing up with some knowledge of food values and of the protective foods; but members of the older generation were often ignorant on those points and were content to appease their appetites with bread and butter and jam and endless cups of tea. The habits of the age had to be respected, and they would not welcome abrupt changes; but experience had shown that much could be done to improve their nutrition by simple explanation, dietary instruction and continued encouragement.

Nutritional Problems in the Pacific.

E. MESSEL (Executive Officer for Health, South Pacific Commission) said that as an epidemiologist and hygienist he had frequently had to consider nutritional problems in the South Pacific; nutritionists would doubtless find his form of approach somewhat unorthodox. It did, however, seem to bring out some interesting aspects. The myth of the Pacific islands where abundant and varied foods fell effortlessly into the hands of surfeited peoples had existed only in the imagination of over-enthusiastic travellers; the real picture was quite different. Today, everyone agreed that on those islands there were qualitative and quantitative nutritional problems, although they did not reach famine proportions. Those problems were still insufficiently known; they had but rarely been assessed; their complete practical solution had still to be found. Several of them had come to light as the result of a few specific surveys carried out in certain territories; elsewhere, superficial observations had led to the advancement of proposals which were much too hypothetical. Study of these data revealed the existence of common problems, some of which were the following: the uncertainties and unknown factors of the nutritive and biological value of diets of which the staples were roots and tubers, often supplemented by fish and sea produce, coconuts and other produce such as nuts, green leaves and so on; inadequate means of preservation and inadequate means of distribution of foodstuffs; and the precarious situation of the particularly vulnerable groups, especially the women and children. Those problems were usually considerably modified by the geological environment (so-called "high" volcanic islands and "low" coral islands), by climatic conditions (seasonal variations), by agricultural and economic conditions (cash crops replacing subsistence agriculture, imports and exports of food products, purchasing power of the peoples *et cetera*), by social conditions (development of the peoples as compared with Western civilization, tribal life, drift towards the towns, standard of education, demographic trends, imports of foreign elements), by local endemic diseases (malaria, intestinal parasitoses) and so on. An accurate assessment of those problems had just begun. Surveys taking account of the various factors influencing nutrition had been very limited and only too often superficial. The staple diets were roughly known qualitatively, and much less well defined quantitatively. The study of the nutritional status itself had just been touched upon by limited and debatable studies of plasma proteins, hæmoglobin, and the mineral components of the plasma or the blood. No metabolic assessment had been made. Notwithstanding that lack of information, the recommendations made on nutrition followed those in use in America or other Western countries.

Dr. Messel went on to say that the surveys carried out had clarified local situations only, at a specific time; a dynamic assessment must replace that static assessment; that meant that efforts had to be made to anticipate the modifications which ceaselessly varying economic and social conditions would make in nutritional problems. For example, it would be wrong to recommend the use of milk and dairy products to the peoples of territories in which there were but distant and uncertain prospects of any developments in cattle breeding, food-preserving industries, transport and distribution, and in which the purchasing power permitted only of occasional resort to imported products. It was not a healthy solution of the problem to propose the use of legumes—soya beans, for example—when the possibilities of growing them and their reception by the peoples were as yet unknown. At the present time, and for many years to come, a hasty general solution by an over-strict application of data collected elsewhere could not be contemplated. Nutritional problems in the Pacific islands were determined by changing conditions and specific aspects; they were local problems which required local assessment and local solutions by a logical use of present possibilities. After a period which could be described as a period of fumbling in that field, the South Pacific Commission, by accepting, at its twelfth session in October, 1953, the recommendations of the Research Council, was now entering on the right road. The present programme was one of dynamic and coordinated approach; every effort would be made to ensure that the physician nutritionist, biochemist, dietitian and food technologist, working in close association with the experts on fisheries and subsistence economics, the sociologists and the educationists, concentrated on the assessment of the problems, clarified their developing trends, and collaborated with the local services, within the framework of their policy, with a view to reaching a reasonable solution.

Nutrition Education in the South Pacific.

D. LANGLEY (South Pacific Health Service) said that education in nutrition had become necessary because recent field work had shown that the diet pattern was changing from one of native-grown foods to one that included store foods, the latter often being unwisely selected. Miss Langley said that it had to be decided what was to be taught and what was the most effective way of teaching it to the islanders. The policy was to teach the autochthonous people to continue using the best of their own cooking practices when they were nutritionally satisfactory. It had become important to advise them on the wise selection of store foods. The change in the food pattern of the native household had had the greatest effect on the nutrition of the infant and the young child. Emphasis was therefore on improved feeding practices. As to the most effective way to teach, it was difficult to find a reason acceptable to the native housewife why good nutrition was important for her family. Teaching was therefore concentrated on those groups receiving extra education, such as school teachers, assistant medical practitioners, nurses and similar groups in the hope that they would teach their own people. Miss Langley described the present teaching programme and showed many of the posters and other educational material that was used. In conclusion, she said that the effectiveness of the teaching programme could be judged only by the standard of nutrition of the next generation of island people. There was a need for improved teaching methods which should arise out of the close cooperation of medical scientists with anthropologists, social scientists and educationists.

Dr. Messel said that education programmes were all very well, but practical demonstrations were very important and should be given again and again in the conditions in which the people lived. He thought that something might be done by women's committees—committees of village women, who might form a nucleus to teach the others.

MRS STILL (Sydney) asked whether the native women were sufficiently literate to follow the meaning of the posters used. She suggested that a practical class might be better than a demonstration.

Miss Langley, in reply, said that the posters were backed up by workers, by nurses and by nutritionists. It was of no use to send posters to the villages. It had not been possible to try the effect of practical classes. A European sister was appointed to each district, and under her worked a team of native trained nurses. At present the nurses had no time to teach cooking—their time was taken up in inspecting babies and mothers. She agreed with Dr. Messel about the formation of women's committees, but thought it would be hard to maintain the interest of a group of village women.

The Food Consumption of Australian Aborigines: Natural versus Civilized Diets.

W. E. WILSON (Canberra) said that the types of food eaten, the customs associated with eating, and the methods of obtaining food were important in any community, not only because of their nutritional implications, but because they formed part of the general culture pattern of the group. The "natural" food of the Australian aboriginal had been gradually supplanted by a diet of foods introduced by white settlers, and the opinion was often expressed that that was a retrograde step which must lead to the decline of the aboriginal race. The aborigines had been nomads, moving camp when the immediate locality was eaten out. They kept no herds and cultivated no crops. A large part of the aboriginal's life had been spent searching for food—the

men hunting the larger animals and fish, while the women and children collected vegetable foods and small animal food such as shellfish, grubs, insects and eggs. The speaker described methods of hunting and gathering foods. The diet was varied, kind and quantity fluctuating according to the area and the season. There was practically nothing alive, animal or vegetable, that the aboriginal would not eat. Every edible part of the food was eaten, and naturally there was little wastage of victuals so hardly won. Foods were roasted on an open fire or in hot sand and ashes or in a stone oven. Miss Wilson described ways of cooking various animal and vegetable foods, emphasizing the fact that little if anything was wasted. The primitive cooking methods would ensure little loss of nutritional value, and the consumption of soft bones, liver, intestines and other organs would also provide important nutrients. Cooked foods were inevitably mixed with a great deal of sand and ash, with the result that the teeth became gradually worn down often to the gum. When food was plentiful all groups would have a variety of foods; at other times the women and children might have had a lesser share of the animal foods. In her report of the nutrition unit in Arnhem Land, McArthur had described the food of four groups living entirely on indigenous foods. Compared with the Recommended Dietary Allowances of the National Research Council, U.S.A., the diets in two of the camps had shown an intake of Calories slightly below normal. The diets had been well balanced.

As to nutritional status, it had been found that the heights and weights of adult aborigines were slightly below those of white Australians. Women appeared to age quickly. Basedow had reported that the teeth of primitive aborigines were strong and well developed and dental caries had been practically unknown; but the teeth were commonly ground down almost to gum level because of the fibrous nature of the diet, the large amount of grit it contained, and the habit of crunching bones and shells. Twenty-four years later Moody had also noted that attrition was an outstanding feature, even among children; on the other hand, he had found dental caries common among older natives. The native dentition appeared to suffer a collapse after the age of thirty to thirty-five years; that had not been confined to mission adherents, but had also appeared amongst the odd small groups of bush natives. He had contended that food impaction due to the extreme coarseness of the diet led to attrition, hypertrophy of the gingival tissue, pyorrhœa and eventually caries. Perhaps after all those primitive natives had not possessed the perfect teeth usually attributed to a "natural" diet.

As to the present-day aboriginal, there was no doubt that the native population had gradually declined; but no accurate statistics were available. The present population was estimated to be 45,000 full bloods and 28,000 part aborigines. Dietary surveys carried out by the Commonwealth Department of Health in 1948 and 1951 had investigated the food of aborigines in Western Australia and the Northern Territory. A few of the natives were nomads, having little or no contact with civilization; a few were "part-time" nomads; some were more or less permanently attached to missions or government settlements; others lived on established cattle or sheep stations; a few lived in individual households near towns, and were for the most part "part-time" aborigines. As to their diet, that of the bush natives would be similar to that of primitive aborigines, except that when food was scarce they would visit settlements where rations were available. Conditions in missions, government settlements and cattle stations varied. The basis of the rations was meat, flour, sugar and tea. In most stations the supply of beef was plentiful; in a few settlements it was meagre. Miss Wilson described variations in diet in detail, and also some of the attempts that had been made to improve them. Summing up, she said that the diets had been graded as A, B, C, D and E, on the basis of the calculated intake of specific nutrients. In the Northern Territory 32 of 38 groups in missions, government settlements and stations had been rated as having poor diets, and in Western Australia all of 21 groups had been rated as D or E grade. In missions and government settlements there had been some attempt to provide some of the foods especially needed by pregnant women and children. Cooking and service on many stations were primitive and did nothing to help the natives assimilate civilized habits. In some government settlements and missions and a small number of stations cooking and service were of better standard.

Despite the fact that their diets were rated low in some nutrients, the aborigines did not show signs of frank deficiency disease. It was also not clear whether their teeth had deteriorated as a result of the consumption of civilized foods. The lack of dramatic evidence of deficiencies should not lead to the easy conclusion that the requirements of aborigines were less than those of Europeans. If the fact was accepted that the aboriginal's "natural" diet was a thing of the past, it was the white people's responsibility to ensure that he had the opportunity of obtaining an adequate diet that was nutritious and acceptable to him, and, as he became assimilated, of a type that would not differentiate him from the white man. For aborigines who were issued with rations, ration scales should be controlled by the appropriate authority; aborigines, particularly the children, who were in a position to choose their own diets should be educated to choose and utilize foods to the best advantage. Now that the declared policy of the native welfare administrators in Australia was the cultural assimilation of the aboriginal, it could be expected that the diet of the aborigines would improve together with their general living conditions, and indeed steps had already been taken in that direction. The complete change of habits and outlook that assimilation involved could not be accomplished overnight or without difficulty; but if it was accepted as a long-range aim, not only by the authorities but by the public, it should be achieved eventually.

World and Australian Food Problems.

A joint session of Sections I and K took the form of a symposium on world and Australian food problems.

The discussion was opened by DR. F. T. WHALEN (Switzerland), Director of the Agricultural Division of the Food and Agricultural Organization in Rome. He gave an outline of world food and agricultural problems, with special reference to staple foods such as wheat and other grains.

J. G. CRAWFORD (Canberra) spoke about Australia's agricultural programme. He said that there was no reason to expect that either Australia or Canada and the United States would face a catastrophe in the world wheat market. Wheat sales from those countries were moving slowly now, but there were good reasons why the demand was not keen just at present. Domestic production in Europe had been well above average last year, and British millers, because of stored supplies, were needing only 50% of their normal demands. A contributing factor to slow wheat sales at present was the uncertainty about the International Wheat Agreement which the United Kingdom had not joined. He suggested that Australia should follow the lead of Canada and the United States and keep larger stocks on hand than in pre-war years. The chairman of the Wheat Board had been fully justified in drawing attention to the present wheat storage problem. Referring to primary production generally, the speaker said that rising costs were a major factor affecting efficiency. One big problem which had to be faced was the production of cheap fertilizers, because already the cost of fertilizers was beginning to worry the Australian farming community. That was an important matter, because it was high time that Australia set about improving the quality of her wheat.

The Contribution of Science to Australia's Food Production Programme.

I. CLUNIES ROSS (Melbourne) said that the contribution of science to Australia's food production had been rather meagre in the past. Australia was producing much less food than Italy. The increase in food production during the eight post-war years had been only 4%. Nothing had been done to explore the deep waters far off the coast as a source of fish supply for the world; Australia must forget any notion that in its on-shore fishing grounds there were resources for a great contribution to world food. On the rural side, the four factors limiting food production were scarcity of water and droughts, poverty of soil, losses from plant and animal diseases, and rabbits. Science had a great contribution to make to irrigation; but not more than a fractional increase in production could be expected from the development of all the potential irrigation schemes now in sight. Dr. Clunies Ross advocated "rain harvesting", which involved conservation in small dams of every drop of water which fell on a property. In the twenty-inch rainfall belt there were thousands of suitable sites for such dams, which could provide invaluable pockets of irrigation. Great progress had been made in improving soils which had been regarded as useless a few years ago. There were in Australia about 15,000,000 acres for which suggestions could be made to overcome deficiencies and bring land into production.

The Influence of Nutritional Findings on the Utilization of the World's Food Production Resources.

F. W. CLEMENTS (Sydney) said that the task of feeding any local group was with few exceptions a problem of local production and distribution. Lord Boyd Orr had written in

1941: "The production and distribution of food must be based on the nutritional needs of the people." That raised the question of what were the nutritional needs of the people. Once they had been determined, the question arose of who should decide the types of foods to be eaten. Sociological as well as nutritional problems were thus involved.

Many attempts had been made to produce lists of nutritional requirements, culminating in the "Recommended Dietary Allowances of the National Research Council of the United States of America" series, the first of which appeared in 1941. Priority and prestige (both national and professional) had given those figures an international status; but it was well to remember that many conditions in the United States in or before 1941 had been conducive to the production of very high standards. Some of those conditions were agricultural surpluses, buoyant economies, recent discoveries of tests to measure tissue saturation for some nutrients, and the fact that these lists of requirements would be used for members of American armed forces. Most of the figures selected had a considerable margin of safety, which might be satisfactory in the United States, but undesirable in countries with depressed economies and inadequate food production. Dr. Clements limited his discussion to a consideration of Calories and protein; adequate Calories ensured satiety, and an adequate supply of protein would ensure the adequacy of a number of other nutrients. Those two—Calories and protein—represented major food production problems.

Dr. Clements went on to discuss the sources of present knowledge of Calorie and protein requirements. He said that some vegetable proteins were now known to have high biological value, and combinations of vegetable proteins could be adequate for maintenance. More recently those combinations had been shown to be adequate for growth as well. Calories could be supplied by wide variations in the percentages of carbohydrates, fats and proteins. The increased use of foodstuffs which were almost pure carbohydrates presented a danger and a challenge. An instance was the use of manioc in place of cereals which supplied protein, minerals and vitamins as well as carbohydrate. As the economic status of an individual or group rose, the tendency was to obtain a higher percentage of Calories from proteins and fats, changes which were seldom made for nutritional reasons. Many peoples in all civilizations habitually ate diets that were nutritionally wasteful and because of their cost were luxury diets. The known facts about diets belonged to the "internal logic" of nutritional sciences. Those facts must be considered in relation to social life, which provided the "external logic". Sociologists now recognized that foods occupied an important place in almost every culture, and that selection of foods by individuals and even by national groups was governed by a hierarchy of values, including cost, health, taste, status or prestige. At present the food patterns of Western civilizations had high prestige. Developments in the nutritional sciences could not be considered in isolation, but must be analysed in the light of current international and local social environments.

Food Technology and Public Health.

A joint meeting of Sections I and B took the form of a symposium on food technology and public health.

Nutrient Additions to Bread.

E. H. HIPSLEY (Canberra) traced the history of policies regarding the addition of nutrients to bread in the United States, in the United Kingdom and in Australia. He discussed the nutritional value of white bread, 60% wholemeal bread and white bread "enriched" to the United States specifications; he said that 60% wholemeal bread was superior to the other two types mentioned. The decreased consumption of meat in Australia since the war indicated that in some diets at least there would be a fall in the niacin and iron intake, unless more wholemeal bread was consumed or unless those two nutrients were added to white bread. Frank deficiency diseases were uncommon in Australia, except amongst alcoholics and people living on "unusual" diets. Although there was very little evidence available on which to base an opinion, it was possible that there might be an appreciable amount of vague ill health which could be prevented by a higher intake of some nutrients. Enriched bread was available in some of the larger capital cities of Australia, where it was sold as a special loaf at a premium price. Under those conditions it could not make any significant contribution to the health of the Australian people; but if it was sold at or slightly above the ordinary price of bread, it might make a significant contribution. The demand for "milk" bread depended on its qualities of texture, toasting and keeping. From a nutritive point of view, the contribution of the added milk was insignificant. The terms "starch reduced" and "protein enriched" bread were misleading and were inaccurate descriptions of the nutrient content of those loaves; those terms should not be used. The term "gluten" bread would be an adequate description.

The Tetrapyrrole Pattern in Nature.[1]

In his presidential address to Section N, R. LEMBERG (Sydney) said that biochemistry and physiology were both borderline subjects between biology and chemistry. As his subject, he had chosen to consider a biologically important class of chemical substances, the tetrapyrroles, from the two polar points of view, theoretical biology and theoretical chemistry, rather than from particular biochemical and physiological aspects. Those two points of view met in the problem of biochemical evolution, a problem which bristled with difficulties, both experimental and theoretical. There palæontology was of no help, and even comparative biochemistry and embryology gave information only at the fringe of the problem. It might be asked why the problem should be raised. Dr. Lemberg said he believed that there were reasons to do so, if only to gain better balanced ideas on the problem of evolution. The morphological and functional levels of organization of living things must be finally correlated with the molecular levels of organization, and the elucidation of that correlation was the ideal task of both biochemistry and physiology.

Biologists tended to assume unlimited possibilities of genetic variations creating increased survival value. That which had survival value would, they believed, emerge sooner or later. External physio-chemical limitations of the environment were, of course, recognized, but not the internal limitations of the underlying physio-chemical mechanism. That was perhaps no serious criticism for the comparatively recent—for example, post-Cambrian—developments. There the enormous complexity of the biochemical organization in each, even the most "primitive" cell known today, made it indeed appear possible that every suitable gene alteration was taken up and responded to by that underlying organization. It was, however, quite another matter to explain the formation of the underlying biochemical mechanism itself. Dr. Lemberg believed that one could assume that extrapolating back in life's history to that early period—a period, for example, before the tetrapyrroles had begun to function—there must have been far fewer possibilities of a gene alteration being picked up by the more primitive level of molecular organization—if such a more primitive level of organization was indeed more than a figment of the imagination. The few scientists who had thought over problems of that early period had so far limited themselves practically to the problem of origin of self-multiplying proteins. The protein pattern was, of course, of particular rank among the chemical patterns of living organisms. Not only was it the genetic carrier of the specificity of species (although the nucleic acid pattern and, in some instances, polysaccharides, might be of equal importance), but it was also the carrier of a manifold chemical specificity. Such patterns might be called α-patterns. Dr. Lemberg believed, however, that it was necessary to pay more attention than hitherto to the problem of evolution of other chemical patterns, which he would call β-patterns—patterns which had little species-specificity, which were found widespread in Nature, often in small amounts, and which constituted the essential "catalysts" of life processes. Among these the tetrapyrroles played a very essential role. The term "catalyst" was not strictly correct. In many instances, substances of that type (for example, tetrapyrroles and iso-alloxazines) formed prosthetic groups of enzymes or coenzymes; but in other instances (for example, hæmoglobin) their activity could not be described as catalytic, or else their mode of action was still unknown. They had been called "ergons". As a third type of pattern one might group the substrates—life's fuel substances and building materials, such as carbohydrates, fats and, again, proteins.

If one wanted to try—it might ultimately prove to be impossible, but try one must—to bridge the enormous gulf between the assumed self-multiplying primitive proteins, supplied by energy from outside, and the organic complexity of the cell, one could not neglect the problem of evolution of the β-patterns or ergons. That held whether one assumed their origin to have been inorganic or biosynthetic.

[1] This address will be published in full in the proceedings of the Congress.

Coming to the tetrapyrrole pattern, Dr. Lemberg said that it was amazing to see the variety of uses to which Nature had put it. Without the tetrapyrroles there would be no photosynthesis and no respiration. In the higher plants and green algæ, photosynthesis required chlorophyll *a* and *b*. In more primitive organisms—for example, the blue and red algæ—bile pigment was found as prosthetic groups of photosynthetic pigments. Bacteriochlorophyll was used in the photoreduction of carbon dioxide in purple bacteria. Cytochrome oxidase and the cytochromes were the catalysts of cellular respiration, and the catalases and peroxidases had similar functions. Hæmoglobin, chlorocruorin and the myoglobins carried oxygen from the outside of bulky animals to the cellular catalysts. With the possible exception of some obligatory anaerobic bacteria and of the viruses, all organisms contained tetrapyrroles, whose amounts in the biosphere measured millions of tons.

Dr. Lemberg said that it might well be asked what made the tetrapyrroles so suitable for those fundamental life tasks. Ease of biosynthesis was an important factor. In contrast to the difficulties which the organic chemist experienced in synthesizing those compounds, biosynthesis proceeded readily in all but a very few organisms. Enormous amounts of tetrapyrroles were synthesized on the earth's surface. The amount of chlorophyll on the earth's surface must be staggering. The amount of hæmoglobin porphyrin synthesized by the human species was close to 160,000 tons of porphyrin (5,000,000 tons of hæmoglobin) annually. That figure was so far below the potential of the synthesis that people could donate a great deal of blood to the Red Cross without detriment. If there were added to that the porphyrin in the hæmoglobins and myohæmoglobins of all other vertebrates and some invertebrates, and the hæmatin enzymes, lower in concentration, but for all that the more widespread, one realized that the process dwarfed all human production of dyestuffs. H. Fischer's synthesis of protoporphyrin in 1929 was rightly considered a triumph; but it was a matter for sober reflection by organic chemists that Fischer's own body produced annually no less than 85 grammes of protoporphyrin for his hæmoglobin alone, more porphyrin than he synthesized chemically in a lifetime.

Dr. Lemberg then considered the physico-chemical basis of the fitness of the tetrapyrroles for their biological function. Colour and light absorption must have been of fundamental evolutionary significance if the oxygen of the atmosphere was mainly produced by photosynthesis. Resonance structure, physico-chemically closely allied with colour, was of importance even where light absorption itself was not. The large central sixteen-membered ring readily accommodated metal atoms to form firm tetrachelate metal complexes—iron in the hæm compounds, magnesium in the chlorophylls. In the hæmoproteins, the porphyrin ring, the iron atom and the protein bound to it, formed an integrated resonance system, in which the activity was centralized in the iron atom, but specificity of action was largely determined by the protein. Finally there was a great possibility of variations on the same theme for Nature to experiment with. The startling progress made in the chemistry of the tetrapyrrole compounds during the first quarter of the twentieth century had been followed by an equally promising development of their biochemistry in the second. The mode of breakdown and biosynthesis of the tetrapyrroles had been elucidated, and it had been shown that their biosynthesis required the preexistence of a large past of intermediary metabolism. Those findings, therefore, raised new difficulties with regard to the problem of biochemical evolution, for the tetrapyrroles now appeared not to be primeval substances, as had often been assumed, but comparatively recent introductions, and with them most of the mode of life as it was known today, based on photosynthesis and respiration. The realm of really primitive organisms was thus greatly narrowed and might be further narrowed by the finding of cytochromes in anaerobes.

Symposia in Section N.

R. LEMBERG (Sydney), commenting on the programme of Section N, said that in contrast to earlier meetings of that section in previous congresses, when a great number of individual papers had been read, some of them good, others not so good, the majority of papers read at the Canberra meeting of Section N were contributions to a few symposia, which had been arranged by the president and the local secretaries. Such symposia dealt with "The Integration of Enzyme Systems", "The Control of Hormone Secretion and Action", "Passage of Ions Through Membranes and Bioelectric Potentials", and "Transmission Across Junctional

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MARCH 6, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	2(2)	..	..	..	..	..	..	2
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	2(2)	..	..	..	..	..	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	11(7)	14(12)	7(6)	..	1(1)	..	2	..	35
Diphtheria	13(8)	..	1	..	2	..	..	..	16
Dysentery (Bacillary)	..	2(2)	1(1)	2(2)	1	..	..	..	6
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	1	9(6)	..	..	3(3)	..	..	..	13
Lead Poisoning	..	4(4)	..	..	..	..	..	..	4
Leprosy	..	..	..	..	..	..	4	..	4
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	5(1)	1(1)	..	..	3	..	..	..	9
Ophthalmia	..	..	..	..	5	..	..	..	5
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	28(15)	10(5)	1	4(4)	39(32)	..	..	5	87
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	7(3)	..	..	12(11)	..	..	..	19
Salmonella Infection	..	..	..	..	2(2)	..	..	..	2
Scarlet Fever	11(4)	13(6)	1(1)	..	5(1)	..	..	..	30
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1(1)	..	..	..	1
Trachoma	..	..	..	..	1	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	44(33)	16(10)	25(7)	10(7)	3(1)	5(2)	1	..	104
Typhoid Fever	..	1(1)	1	..	..	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	1(1)	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Regions in the Central Nervous System". Of those the first had been particularly successful and of a standard equal to the world's best, partly because of the active contribution by Australian biochemists to research in that field, and partly because that field was now ripe for integration. Dr. Lemberg said that there was often quite silly criticism of over-specialization; such critics forgot that intense analytical penetration in great detail was necessary for integration on a more advanced level. The field of enzyme biochemistry had just reached the point where such reintegration appeared possible.

General Council Meeting.

The formal business of the thirtieth meeting ended with a general council meeting held on January 20 at the Australian National University, Canberra. After some discussion it was agreed that the next meeting of the Association should be held at Melbourne in August, 1956.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

Programme for April.

Classes for Part I of Higher Qualifications.

Classes for candidates for Part I of higher qualifications which commenced at the University of Melbourne in March will be continued in April.

Country Courses.

Sale.

On April 3 and 4 a week-end refresher course will be held at Sale with the following programme: Saturday: 2.30 p.m., Dr. W. E. King, "Recent Advances in Thyroid Disease"; 8 p.m., Dr. R. Officer, "Intestinal Obstruction". Sunday: 10.30 a.m., Dr. H. B. Hattam, "Stress Incontinence in the Female". The fee for attendance is at the rate of 15s. per lecture, but those who have paid an annual subscription to the committee are invited to attend without further charge. Inquiries should be addressed to Dr. J. M. Gooch, Cunninghame Street, Sale.

Flinders Naval Depot.

On April 14, at 2.30 p.m., by arrangement with the Royal Australian Navy, a demonstration will be given at Flinders Naval Depot by Dr. Keith Bottomley on "Hypertension".

Courses for Part II of Higher Qualifications.

M.D. Part II and M.R.A.C.P.

A course suitable for candidates for M.D. Part II and M.R.A.C.P. will be held in June and July. It will be conducted by the honorary staff of the Alfred Hospital and will extend for forty mornings over eight consecutive weeks and consist of eighty sessions of one and a half hours. The course is designed primarily as a preparation for examination and is not necessarily to be regarded as an advanced post-graduate course in various medical specialties.

Sessional time of clinical examinations and demonstrations will be concerned with actual trial examination of candidates on unknown cases. Over thirty members of the hospital staff will conduct the classes and distribution of sessional times is approximately as follows: cardiology (eleven), pulmonary diseases (seven), alimentary diseases (six), neurology (six), endocrine diseases (five), hæmatology (five), renal diseases (four), clinical examinations (ten), radiology (four), macroscopic pathology (four), clinical pathology (two), medical ophthalmology (two), dermatology (two), electrolytes (two), allergy (two), psychiatry (two), skeletal disorders (two), infections (one), antibiotics (one), anæsthetics (one) and obscure disorders (one).

Enrolments should be placed by April 30 with the Melbourne Permanent Post-Graduate Committee at 394 Albert Street, East Melbourne. The Committee will also be pleased to answer inquiries. The fee for the course is £31 10s.

Ophthalmology.

From May 3 to September 30 the Victorian Section of the Ophthalmological Society of Australia (British Medical Association) will conduct a course of over seventy sessions on ophthalmology, including special pathology, for candidates for the D.O. Part II. Sessions will be held in the late afternoons, chiefly on Mondays, Thursdays and Fridays. They will consist of lectures, clinical demonstrations at various hospitals and pathology demonstrations at the Eye and Ear Hospital and the University of Melbourne. The fee for the course is £31 10s.

Enrolments should be placed by April 15 with the Melbourne Permanent Post-Graduate Committee at 394 Albert Street, East Melbourne, from whom inquiries may be made.

Other Courses.

Radiodiagnosis and psychiatry courses will commence in late May or early June, and announcements will be made later. Bacteriology classes will commence in June.

Diary for the Month.

April 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
April 7.—Victorian Branch, B.M.A.: Branch Meeting.
April 7.—Western Australian Branch, B.M.A.: Council Meeting.
April 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to the Editor, The Medical Journal of Australia, *The Printing House, Seamer* Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, *The Medical Journal of Australia, Seamer Street, Glebe,* New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by *applying to the Manager or through the usual agents and booksellers.* Subscriptions can commence at the beginning of any *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.